COUNTWAY LIBRARY
HC 18F4 E

Library
Boston Psychopathic
Hospital

Boston, Massachusetts

PROPERTY
MASS. COMMISSION
ON MENTAL DISEASES
PATHOLOGIST'S OFFICE.

MEDICAL DISEASES OF THE WAR

Digitized by the Internet Archive
in 2025

https://archive.org/details/medicaldiseaseso0000arth

MEDICAL DISEASES OF THE WAR

BY

ARTHUR F. HURST, M.A., M.D.(Oxon.), F.R.C.P.

TEMPORARY MAJOR, R.A.M.C.; PHYSICIAN AND NEUROLOGIST TO GUY'S HOSPITAL; NEUROLOGIST TO THE ROYAL VICTORIA HOSPITAL, NETLEY; LATELY MEMBER OF THE MEDICAL ADVISORY COMMITTEE, MEDITERRANEAN EXPEDITIONARY FORCE, AND CONSULTING PHYSICIAN TO THE SALONICA ARMY

SECOND IMPRESSION

BOSTON MEDICAL LIBRARY
IN THE
FRANCIS A. COUNTWAY
LIBRARY OF MEDICINE

LONDON
EDWARD ARNOLD
1917

[*All rights reserved*]

33A
H943
646

PREFACE

I HAVE had constant opportunities of studying every phase of the various diseases occurring on active service, first as physician and neurologist to a number of military hospitals in London and to the New Zealand Hospital at Walton-on-Thames, then as a member of the Medical Advisory Committee for the Prevention of Epidemic Disease in the Mediterranean Expeditionary Force in Lemnos, and subsequently in Salonica, where I also acted as Consulting Physician to the British Forces.

This book is a record of my own observations, amplified by a study of the literature so far as time and opportunity have allowed. I have read most of the papers bearing on the subjects dealt with, which have been published in the English medical journals, together with a number of the more important French contributions and a small number of those which have appeared in the German language. The principal references are given at the end of each chapter.

I should like to add, for the benefit of those who are more familiar with my former name of Hertz than with my new name of Hurst, that I have made the change because under present conditions it is natural for one of English birth and English descent for several generations to be unwilling to retain a German name.

ARTHUR F. HURST.

November, 1916.

CONTENTS

CHAPTER		PAGE
I.	Functional Nervous Disorders	1
II.	Dysentery	41
	(i) Amœbic Dysentery	41
	(ii) Bacillary Dysentery	52
	(iii) Flagellate Diarrhœa and Dysentery	60
	(iv) Ciliate Dysentery	61
	Note on the Treatment of Diarrhœa during Dysentery Epidemics	62
III.	Amœbic Hepatitis and Hepatic Abscess	64
IV.	Trench Fevers	71
V.	Paratyphoid Fever	88
VI.	Epidemic Jaundice	104
VII.	A Form of Beri-beri occurring among British Troops	116
VIII.	Soldier's Heart	126
IX.	War Nephritis	135
X.	Gas-poisoning	142
	Index	149

MEDICAL DISEASES OF THE WAR

CHAPTER I

FUNCTIONAL NERVOUS DISORDERS

Functional nervous disorders have been relatively more common in this than in any previous war, mainly owing to the use of enormous numbers of high-explosive shells. The symptoms produced do not differ from those seen in civil life, but they have been common instead of rare. It is difficult to classify them satisfactorily. They might, for example, be separated under the heads of neurasthenia, psychasthenia, and hysteria, but these conditions are often present together, and many cases occur which cannot be correctly included under any of these heads. They might also be classified according to their etiology; the largest proportion of functional nervous disorders are due to the effects—apart from actual wounds—produced by high-explosive shells, whilst others result from the long continued physical and mental strain caused by active service; a single experience of exceptional horror is often sufficient, such as seeing a friend killed whilst talking with him, or being in a trench subjected to the prolonged and continuous shelling which precedes an attack. A horrible sight may be completely obliterated from the memory only to return in dreams, or it may be voluntarily repressed by the patient, who is unwilling to talk about it and becomes emotional when he does so. The symptoms produced in these different ways may, however, be identical. No scientific classification will therefore be attempted, but the various functional nervous disorders, which have been observed during the war, will be successively described after a brief discussion of the etiology of neurasthenia and the pathogenesis of shell-shock.

Etiology of Neurasthenia in Soldiers

Neurasthenia or nerve exhaustion has been exceedingly common, especially among men who have suffered exceptional hardships, such as soldiers in the original Expeditionary Force, who were in the retreat from Mons, and those who survived the landing at Gallipoli to spend several months on the Peninsula, but it would require a very exceptional nervous system to pass through twelve months or more of war under modern conditions without exhaustion, even in the absence of shock due to the explosion of shells or to actual wounds. The liability to neurasthenia is greatly increased, if a man's nervous system was in a depressed and irritable condition before he went on active service or if he had previously suffered from neurasthenia.

In all cases the primary factors have been physical fatigue and mental strain. For this reason officers have suffered more than men, as they have been exposed to equal fatigue, but have had more mental strain, especially when unexpected responsibilities have fallen upon young officers, whose seniors have been killed or wounded. In many cases there is also a toxic factor; toxæmia due to diarrhœa, trench fever, or other infection, which is not sufficiently severe to cause a man to go into hospital, is particularly likely to cause nervous exhaustion. A subaltern in the Flying Corps, who was actively engaged almost daily from September, 1914, appeared to have passed unscathed through all the fatigue and excitement until January, 1915, when he had an attack of acute tonsillitis. He was taken to a hospital, where there was a shortage of food, and he was then conveyed in a half-starved condition to England, the journey occupying over thirty hours. The fatigue and insufficient food were not new experiences, but the addition of the intoxication caused by the tonsillitis, which by itself would have only led to a few days of illness, was sufficient to cause a condition of severe nervous exhaustion, the recovery from which was, however, remarkably rapid. In the early months of the war I saw several cases of neurasthenia following anti-typhoid inoculation, when the patient had to march a long distance immediately afterwards. Fortunately the authorities subsequently issued an order that all inoculated men should be off duty for forty-eight hours.

There is no doubt that great heat increases the liability to neurasthenia in a man exposed to mental and physical overwork and to toxæmia.

A very important group of cases is that in which an injury is the exciting cause. Every wound is likely to result in neurasthenia, if the soldier is already over-fatigued from mental and physical strain; the physical shock at the moment, combined with pain, loss of blood and septic poisoning are likely to depress the strongest nervous system. The traumatic neurasthenia, in which no actual wound is present, but which is due to the concussion caused by the bursting of a high-explosive shell in the immediate neighbourhood, forms part of the clinical picture of "shell-shock."

Pathogenesis of Shell-shock

The symptoms produced by exposure to the forces generated by the explosion of powerful shells without any visible injury may be grouped under the head of shell-shock. In many cases physical concussion has also occurred; the patient may be struck or buried by the sand-bags of a falling parapet, or he may be blown into the air and fall heavily on his head, although no bruise can be found.

Symptoms occur most readily in men with a neuropathic inheritance, and in those who are abnormally emotional or have previously suffered from a "nervous breakdown" or concussion as a result of injury to the head. Men with a good family history, who have never suffered from any nervous disability, are less liable to shell-shock; a single explosion is unlikely to give rise to symptoms unless they are already weakened by a long period of physical fatigue combined with severe mental strain, except in the case of explosions of exceptional severity, such as those caused by "Jack Johnsons;" more frequently serious symptoms only develop after the third or fourth explosion.

Several French observers have observed that if a lumbar puncture is performed in a case of shell-shock within a few hours of the onset of symptoms, the cerebro-spinal fluid is generally under increased pressure and contains albumin, blood, and slight excess of lymphocytes. If the examination is repeated in forty-eight hours no abnormalities are found. This accounts for the fact that the cerebro-spinal fluid is almost invariably normal when

the lumbar puncture is performed at a base hospital. Roselle and Oberthur examined a number of men actually in the trenches immediately after they had been blown up. The tendon reflexes were exaggerated and the cutaneous reflexes absent, except the plantar reflex, which was extensor; extreme hypotonus of all muscles was present. In many cases fæces and urine was passed involuntarily, but retention sometimes occurred at a later stage. All these symptoms disappeared in a few hours, often whilst the patient was still unconscious. In such cases it is clear that organic changes have occurred in the central nervous system, which are, however, so slight—consisting probably of minute capillary hæmorrhages and chromatolysis of nerve cells—that they rapidly and completely disappear. The symptoms had thus originally an organic basis, but their persistence and exaggeration are due to psychical causes. Though primarily organic, they are ultimately functional. In some cases the slight organic lesions may persist; the recovery which follows suitable treatment is then only partial. In other cases the original changes in the central nervous system are more severe, although there may be no visible injury; the symptoms may then be organic in origin throughout, and in exceptional cases the injury may be fatal. No definite line can be drawn between the cases in which the symptoms are obviously organic and those in which they are obviously hysterical, as many intermediate mixed cases occur, and the obviously hysterical cases are indistinguishable from those in which an early examination has shown changes in the cerebro-spinal fluid or definite symptoms of organic disease, which completely disappear within a few hours or days of the onset.

There can be no doubt that psychical factors are of great importance in almost all cases, but these observations show that they are not as a rule the only factors, and that it is wrong to regard the physical concussion resulting from a shell explosion "as an extremely rare and unusual cause" of shell-shock. Wiltshire was led to this conclusion by his remarkable observation on British soldiers, which agrees with that of Meige on French soldiers, that it is very rare to find symptoms of shell-shock among the wounded, who are indeed wonderfully cheerful, although they must have been exposed to the conditions which induce it to a greater extent than the unwounded, among whom the

large majority of cases occur. I do not, however, agree that functional neuroses are rare among the wounded, as I have seen large numbers of cases in which hysterical symptoms of various kinds were associated with wounds, the onset of the symptoms being due to circumstances connected with their infliction.

The aerial compression generated at the moment of detonation of a high-explosive shell may amount to ten tons to the square yard. This may be transmitted through the cerebro-spinal fluid to all the neurones of the central nervous system, the concussion of which results in temporary loss of function, due probably to slight and temporary, but none the less definite changes in the central nervous system.

In every battle enormous quantities of carbon-monoxide are produced from cordite. The diffusive power of the atmosphere is so great that the concentration of the gas under ordinary conditions is never likely to reach 1 per cent., which is the strength necessary to cause unconsciousness after a very short exposure. But Mott has pointed out that if a man is buried under sand-bags in a heavily shelled trench or dug-out, or if for any other reason he is confined in a small space, in which the gas produced by an explosion collects without being able to diffuse rapidly away, he will be rendered unconscious, if he is not already unconscious as a result of concussion. The continued inhalation of the gas will cause him to suffer from the effects of carbon-monoxide poisoning, although he will not have realised during his period of consciousness that he was inhaling a poison, as the gas is odourless. Carbon-monoxide poisoning leads to deficient oxygenation of the blood owing to the gas combining with some of the hæmoglobin, which has a more powerful affinity for it than for oxygen. The symptoms produced by carbon-monoxide poisoning in civil practice are headache, mental confusion, loss of memory, inability to concentrate, loss of speech, and visual hallucinations, all of which frequently occur in shell-shock. Post-mortem examinations after carbon-monoxide poisoning show multiple punctate hæmorrhages in the white matter of the brain, especially the corpus callosum, internal capsule and cerebral peduncles, and the nerve cells, especially of the respiratory and cardiac nuclei of the medulla, show chromatolysis with eccentric nuclei. These changes are identical with those found in men who die without regaining

consciousness after being blown up and buried by high-explosive shells in the absence of any external injury.

The terror caused by the explosion, in the few moments which elapse before consciousness is lost, is probably an important factor in the production of the hysterical and mental symptoms which form part of the picture in many cases of shell-shock, as they do not develop if the explosion occurs whilst a man is asleep. In some cases past experiences, which were associated with powerful emotions but which may in the course of time have been almost forgotten, are re-awakened by recent terrible events and give colour to some of the symptoms.

The symptoms of shell-shock are very varied, and it is at first difficult to explain why one man should become blind, another deaf and dumb, and another hemiplegic under apparently identical conditions. From careful inquiry into the history of numerous cases I have come to the conclusion that the variability of the symptoms can best be explained in the following way. As a result of the various factors which give rise to shell-shock all the functions of the body are in abeyance for a period which varies in different cases: the patient cannot see or hear or feel, he cannot talk, he cannot move, and his mind is a complete blank. In many cases all these lost functions gradually return in the course of a few hours, though some come back more rapidly than others: the patient is then in much the same condition as a man who has been concussed by falling on his head—with a number of symptoms, such as headache, difficulty in concentration, and lack of energy, but there is no paralysis nor loss of any of the special senses. In other cases, as consciousness returns, the patient's mind becomes fixed on some part of his body which is painful, the pain being the first impression powerful enough to attract his awakening attention; or the temporary inability to see or hear or speak, which generally remains unnoticed when consciousness first returns because of the absence of any desire to see or hear or speak, is suddenly realised owing to a special call being made on one of these functions. The patient's dawning intelligence becomes fixed upon this single missing function, and he suggests to himself that the disability will be permanent. The fact that the other functions are missing remains unnoticed, and after a time they spontaneously return. The persistent localised loss of function is thus caused by

auto-suggestion leading to the perpetuation of what would otherwise be a very temporary incapacity: it can thus correctly be described as hysterical. Less frequently the impressions received by the patient between the explosion and the loss of consciousness give the key to the symptoms. The first thought on regaining consciousness of a man deafened by the noise, speechless with terror or struck in the back by a sand-bag when the explosion took place, may result in the suggestion of deafness, dumbness, or pain in the back and paraplegia.

The first case of hysterical amblyopia described on page 15 affords a good example of this chain of events. The patient was blown up by a shell which exploded on the sand-bags in front of him, sand being thrown into his eyes. His first impression on regaining consciousness was extreme irritation in his eyes. He tried to open them, but found he could not do so. His mind thus became concentrated on his eyes, and owing to the confusion, which is common among the uneducated between inability to open the eyes and blindness, he became obsessed with the idea that he was blind and that he would never be able to open his eyes or see again. The impairment of other functions, which was doubtless present at first, remained unnoticed in this greater trouble, except for loss of hearing, which proved quite temporary. The inability to open the eyes and the idea of blindness were thus perpetuated by auto-suggestion, and persisted long after the inflammation caused by the dust had disappeared. The second and third cases had a similar origin, the result of auto-suggestion being aggravated in the latter by unintentional suggestion on the part of the medical officer. The fourth and sixth cases illustrate the principle that a part of the body, the functions of which were previously below normal, recovers less rapidly than the other parts; an error in refraction, which was previously ignored, increased the loss of function caused by shell-shock, so that the patient's attention was drawn to his eyes and by a process of auto-suggestion the loss of vision was perpetuated.

The first impression which reached the returning consciousness of another man who had been blown up by a shell was a sense of extreme thirst. He found he could make no sound when he tried to call for water: the inability to speak came as a shock to his mind, and the fact that all the other functions of his body were

also more or less in abeyance remained unnoticed. With his mind fixed on his dumbness, it was impossible for the power to speak to return when his other faculties came back. Strong counter-suggestion overcame the fixed idea of mutism and recovery took place.

A man was blown up by a high-explosive shell and fell on his right shoulder. He told me that his first impression when he regained consciousness was of pain in the shoulder, and in order to relieve it he tried to move his arm. He found he could not do so owing to the loss of power of movement caused by the concussion of his central nervous system—a loss of which he would never have become aware if the pain had not made him wish to move, as in his semi-conscious condition he would have been content to lie inert. His mind thus drawn to his inability to move his arm became obsessed with the idea that it was paralysed, and when the power to move and the other functions of his body returned, the right arm remained paralysed, although the pain quickly disappeared. The hysterical monoplegia in this case was cured by suggestion.

Symptoms

Stupor.—The immediate effect of a high-explosive shell is to render a man unconscious. In the severest cases the patient's breathing is stertorous and he may die after an interval of a few hours or days without regaining consciousness. In the more serious of the cases in which recovery ultimately takes place the patient passes into a condition of stupor. He remains for a time entirely unconscious of his surroundings. He cannot see, hear, or speak, and pinching his skin produces no response. The reaction of the pupils to light is impaired or lost. Micturition and deglutition are unaffected. The limbs are generally very rigid, the thighs being flexed on the trunk and the hands clenched. The patient sometimes appears to live again through a past experience, most probably the one which immediately preceded the stupor. In a striking case related by Aldren Turner the man lay curled up under the bedclothes. From time to time he would look out, as if peering over the parapet of a trench, stare wildly around him, and then hide under the clothes. These actions were constantly repeated for

several days with gradually diminishing frequency. Mott describes the case of a corporal who was sent out with some men to repair the barbed wire in front of the trenches. A big shell burst and blew him some distance into a hole. He scrambled out and saw his comrades lying killed and wounded around him. He then lost consciousness, and when first seen in London some days later he appeared to be in abject terror, constantly muttering "Dead all round." The expression of terror disappeared when normal sleep returned.

In severe cases complete insensibility is soon replaced by a dazed condition, in which automatic complex acts may be performed. A man may be found several miles away from where he was blown up, but he will never recall how he covered the distance.

The duration of the stupor varies from a few hours to several days. Sometimes the blindness and deafness only last a few minutes after the return of consciousness. Vision generally returns first and hearing shortly afterwards, the patient remaining mute for a considerably longer period. This is, however, not always the case, as hysterical blindness or hysterical deafness with or without mutism may persist alone long after the other symptoms have disappeared. The very temporary loss of the other functions may appear so trivial to the patient compared with the persistent symptom that he may not mention it, even if he remembers it, unless special inquiry is made. Cutaneous and deep anæsthesia quickly disappear and may be replaced by hyperæsthesia; the patient is sometimes frightened whenever he is touched, especially if he is blind and deaf and cutaneous sensibility remains his only protective sensation.

The stupor may suddenly pass away, the patient having no recollection of what occurred between the onset and the moment of recovery. More frequently improvement is gradual. Some patients remain lethargic for a long time and take no interest in what is happening around them, but they may obey simple commands, such as to put out the tongue, but only after a considerable latent period.

Amnesia.—As a rule the patient soon recovers his memory up to the time of the explosion and may even recollect that he heard the sound of the shell coming, but from this moment his mind is

a blank. Less frequently the memory is perfect up to a certain date, such as the day of arrival at the front, but all subsequent events are forgotten; the patient may live over some of the forgotten events—especially those of a terrifying nature—in his dreams, but on waking he remembers nothing of them. In severe cases, especially if the shell-shock was accompanied by concussion or burial, more or less complete retrograde amnesia may be present. In two cases of this kind memory only began to return after I had induced the patient to write his name and other details during hypnotic sleep. In the worst cases the patient's mind is a complete blank; he looks dazed and bewildered, he does not remember his name nor recognise his relations, and he has no recollection of any past events and immediately forgets everything he is told.

Sooner or later memory begins to return. This may be hastened by anything which tends to recall a man's previous occupation: McDougall relates the case of a man, who showed great aptitude for the task when given a pair of scissors and told to cut the hair of another patient; he said that the occupation seemed familiar, and it eventually turned out that he had been a barber. A man who is fond of music may remember tunes and words of songs, especially if they are begun for him, long before he remembers anything else; this is due to the fact that the singing of a familiar tune can normally be continued without any effort of consciousness after it is once started, knowledge which appeals particularly to the emotions, such as music, being most deeply engraved in the mind.

Double Personality.—Feiling records the case of a young soldier who was buried in a trench for fifteen hours in October, 1914. When he came to himself five days later he found that he was in Manchester, but he had lost all recollection of his previous life and had to learn to speak, read, and write again; he did this so quickly that at the end of January he was normal in every way, except that he had forgotten all events up to his re-awakening in Manchester. He did not recognise his parents or any other people or objects with which he had previously been familiar. When hypnotised he could answer questions about his previous life, giving vivid details of his experiences in Flanders. He wrote answers

to questions whilst under hypnosis, but when he woke up he did not recognise the handwriting, which was quite different to that which he had since acquired. When hypnotised he believes that it is November, 1914, and that he has just arrived in Manchester from the front : he recognises his father with a degree of affection, which is quite absent from his usual state, as when awake he has to take him on trust. So far all attempts to associate the hypnotic with the waking state have been unsuccessful.

Headache.—Headache is invariably present ; whilst the patient is still dazed as a result of the shell-shock, his head feels heavy and uncomfortable, but severe pain only develops as his mind becomes clearer. It is increased by the smallest mental effort, and is often worst at night, when it may prevent sleep. Lumbar puncture often shows that the pressure is raised, and the removal of cerebro-spinal fluid then relieves the headache. It is greatly aggravated by nightmares and the recollection of horrors through which the patient has passed. It varies in character and position, but it is most often in the occipital region and back of the neck. For many months after the severe headache has disappeared, a heavy, full sensation may still be caused by mental concentration or excitement, and sustained attention is impossible because of the sense of weariness it causes. The patient finds it difficult to make up his mind even about trifling affairs ; this and the inability to use his brain for any length of time are often a great source of worry.

Mental Irritability.—Mental irritability is very common, especially among officers and the better educated men. It is an early symptom and often continues after the patient is otherwise well. He loses his temper for trivial causes and may get himself into trouble for insubordination.

Fatigue.—A feeling of great fatigue is the most constant symptom of neurasthenia and is often present in shell-shock. The patient is unable to exert himself either physically or mentally. In severe cases he likes to lie like a log in perfect silence and a darkened room with his mind completely blank ; it is only when improvement begins that he desires cheerful surroundings, but even then thinking requires an unpleasant effort.

Insomnia, Nightmares and Hallucinations.—Insomnia is very common, and many men live through their most terrifying experiences night after night in their dreams. One officer was found every morning sleeping on the floor, as he invariably dreamt that he was fighting on the parapet, which was represented by his bed, and that he finally saved himself by tumbling into the trench, which was represented by the floor. Occasionally experiences at the front are blended with episodes which occurred many years before and made a profound impression on the patient's mind. In some cases the dream is forgotten, the patient only recollecting that he woke with a start and found himself in a cold sweat.

In severe cases the patient constantly thinks of his horrible experiences whilst awake, and he may even have hallucinations, which occasionally result in insane conduct. The terrified aspect and the constant tremor, rapid pulse and profuse sweating, observed in the worst cases for many weeks after the onset of symptoms, are doubtless the result of the mind remaining fixed on past experiences.

A nineteen-year-old soldier saw an old woman's head blown off by a shell whilst he was standing near her in front of her cottage. The episode constantly appeared as a nightmare, which prevented him from getting any real rest, and hardly an hour passed in the day in which the scene was not vividly recalled to his mind. The want of refreshing sleep at night and the disturbing thoughts during the day made it impossible for him to recover from the neurasthenia, which had resulted from the exhausting experiences he had passed through. Suggestion under hypnosis repeated on three occasions resulted in the disappearance of the nightmares and of the constantly recurring recollection of the horrible scene he had witnessed, and his general condition consequently improved so rapidly that he was able to return to duty a month later.

The circulatory symptoms described in the chapter on the soldier's heart (p. 124) are often present, palpitation being particularly troublesome in some cases. The pulse is often fast, but the blood-pressure is rarely raised and is often remarkably low.

It is a curious fact that the appetite is generally excellent and the digestion is good, though constipation is common, the absence of severe digestive symptoms being apparently a peculiarity of war neurasthenia.

Obsessions.—I have seen a few soldiers, in whom agoraphobia or dread of open spaces, was well marked. A striking case of an obsession was that of a captain, who with one lieutenant was the sole survivor among the officers of his battalion at the battle of Ypres. He received the D.S.O. for his gallant conduct on that occasion, but the enormous and unexpected responsibility, which devolved upon him when he was left in command, was too much for him in his physically exhausted condition. Though he had saved the remnant of his battalion through his own almost unaided efforts, he felt that he could never face any responsibility again and that the next occasion he was in danger he would have so little control over himself and his men that he would disgrace himself. This obsession took complete hold of him, and he became more and more depressed in consequence. When, after many weeks spent in the trenches, he heard that there would be an attack at Neuve Chapelle the following day, he broke down completely, but with a supreme effort he got through the first day of the battle. Though he did very well, he was more miserable than ever in the evening and felt he could not face another day's fighting. He was invalided home and arrived in a condition of profound exhaustion and utter misery, as he thought he had disgraced himself for ever. His appetite remained good, but his nights were disturbed by bad dreams. The treatment for such a case is the same as for neurasthenia, but complete rest alone would be insufficient to restore the patient's confidence in himself. Suggestion in the hypnotic state was clearly indicated, and was followed by such rapid improvement that the patient was soon able to return to duty.

Ocular Symptoms

Functional Asthenopia.—The important part played by asthenopia in the production of neurasthenic symptoms has long been recognised. Harwood has recently drawn attention to its importance in shell-shock. In both conditions the activity of the ciliary muscles is impaired, like that of the other muscles of the body. In severe cases of shell-shock the patient may be unable to read for weeks owing to complete inability to accommodate. But even a slight degree of asthenopia is important if there is an error of refraction, although this may never before have caused any trouble. At first light is intolerable and the patient makes no

effort to use his eyes, but after a time he begins to look at his surroundings, and this alone may be sufficient to cause headache and giddiness. Later he tries to read; the difficulties of near vision are now added to those of distant vision, and the neurasthenic symptoms, especially headache, become aggravated.

The refraction of all soldiers suffering from headache as a result of neurasthenia or shell-shock should therefore be tested, if rapid improvement does not occur; when there is reason to suppose that asthenopia is an important factor, the eyes should be examined under atropine whilst the patient is still in bed. Glasses must be worn all day at first; they should be given up for distant vision as soon as possible, but should be used for near vision until complete recovery has occurred.

Hysterical Blindness.—Whilst a man is still semi-conscious or dazed as a result of shell-shock he is often completely blind. Rapid improvement generally occurs, the majority of cases recovering within a week. Occasionally, however, the blindness persists and in severe cases would apparently remain permanent if not properly treated. The eyes are kept closed, and the lids frequently "flutter" or "twinkle," even in a subdued light. On attempting to force the lids open the patient resists by contracting his orbicularis muscle; when this is overcome to a sufficient extent to see the globes, they are found to be directed as far upwards as possible, and the patient is unable to look downwards. The pupils react normally to light, and the optic discs show no change. The patient complains of pain and photophobia, and is much fatigued after being examined. He does not move about like a blind man, but avoids hurting himself, though he does not relax the groping action of people with extremely defective sight. Judged by every test, he is psychically blind, and there is no difficulty in differentiating him from a malingerer, as he passes through long periods of real mental distress. Any lack of recognition of the functional nature of the condition in the early stages greatly prejudices the prognosis.

Early in the war the cases were treated by rest, tonics, punishments, such as abstention from tobacco, confinement to bed or in isolation rooms, persuasion, encouragement and counter-irritation; but all these means were comparatively ineffective,

except in the slighter cases which seemed to recover equally well if left to themselves. In September, 1915, Captain A. W. Ormond asked me to see a number of cases with him, and we decided to try hypnotism.

Case 1.—The patient, aged 22, was looking over a parapet on July 18th, 1915, when a shell struck the sand-bags in front of him. He remembers the sand being thrown up into his eyes, after which he fell back and knocked his head. He was rendered unconscious for twenty-four hours. When he regained consciousness he found that he was completely blind, except that he could just distinguish light from darkness with his left eye. His eyes felt very sore, and his eyelids were constantly quivering; he was partially deaf and had a severe headache. His hearing soon returned, and the headache rapidly improved, but the condition of his eyes had not altered when I first saw him with Captain Ormond on September 17th. He was quite blind, and there was a constant flicker of his eyelids, which were kept almost closed. On forcibly opening his eyes they were found to be turned so far upwards that it was difficult to see even the iris. A few fragments of sand were still embedded in the conjunctiva but not in the cornea; there was no inflammation present.

The patient was easily hypnotised, and whilst asleep he was told that he would be able to see when he woke up. The moment he awoke the suggestion was repeated very forcibly, and his eyes were held open. He cried out that he could see, tears ran down his cheeks, and he fell on his knees in gratitude, as he had thought that he was permanently blind and believed that his sight had been restored by a miracle. When seen again on September 20th the external appearance of his eyes was normal, and he said that he was able to see as well as he had ever done. Captain Ormond, however, found that there was some opacity of the vitreous of the left eye, secondary to hæmorrhage from a retinal vessel; this was doubtless a result of injury at the time of the explosion. There had been no return of symptoms, and the patient was well in every way when I last saw him on September 30th. His vision was 6/6 in the right eye and 6/36 in the left.

Case 2.—The patient had an attack of blindness following conjunctivitis, which was caused by a sandstorm in Egypt early in 1915. He recovered from this in ten weeks, and six weeks later went to the Dardanelles. On July 12th a shell struck a sand-bag immediately in front of him and the sand flew into his

eyes. He did not lose consciousness, but his sight gradually became more and more deficient until at the end of ten days he was only just able to distinguish light from darkness. Very slight improvement occurred spontaneously, but his condition when seen on September 17th was identical with that of the first case, a few pieces of grit being still embedded in the conjunctiva, although there was no inflammation.

The patient was easily hypnotised, but there was very little improvement at the moment. Considerable improvement, however, occurred during the next three days. He was hypnotised on three more occasions; he could see perfectly well on September 30th, but still had some photophobia and wore dark glasses, as he constantly blinked when they were removed. He was therefore hypnotised yet again, and it was suggested to him that the photophobia and blinking would now cease. The result was completely successful, as all symptoms had disappeared by October 4th, although no attempt had been made to remove the grit from the conjunctiva.

Case 3.—The patient, aged 20, was rendered unconscious for a few minutes as a result of the explosion of a shell near him on August 21st in the Dardanelles. Some of the powder was blown into his eyes, which were very sore when he regained consciousness, although he was still able to see quite well. During the next twenty-four hours his vision became more and more impaired. The powder was removed from his eyes when he was taken on board the hospital ship, and his eyes were bandaged. After a few days he thought he would be able to see quite well if the bandages were removed, but the medical officer in charge told him it would be dangerous to do this. During the voyage home he was not allowed to remove the bandages, and he became more and more convinced that this must be because the medical officer thought he was blind. The bandages had not been removed when he was admitted into the hospital on September 25th. On removing them he was found to be in exactly the same condition as the preceding patients. He could distinguish light from darkness, but was unable to see anything, and he kept his eyes turned up and his eyelids closed and constantly twitching.

On September 27th he was hypnotised by Captain Ormond, after which he found that he could see quite well, but the light still worried him and the blinking continued, though to a diminished extent. He rapidly recovered, and was soon able to return to duty.

Case 4.—The patient, aged 29, was knocked over by a high-explosive shell in the Dardanelles and remained unconscious

for a considerable time. On coming round he found he could only distinguish light from darkness; there was no smarting of the eyes, but he constantly blinked. He had a slight headache, but was otherwise well. He began to improve about September 10th, so that he could recognise shadows passing in front of his eyes, but no further improvement occurred until he was hypnotised on September 18th. There was very slight improvement as the result of suggestion at the time, but when seen again on the 20th he said that he was beginning to recognise objects, and the blinking was less marked than before, but he still kept his eyeballs turned upwards and his eyes almost closed. He was hypnotised again on September 20th and 25th, and when seen on the 30th his sight was quite normal and the blinking had completely ceased. Subsequent retinoscopic examination revealed the presence of a considerable degree of myopic astigmatism in the right eye and mixed astigmatism in the left.

Case 5.—The patient was signalling from a gun limber on April 28th when he was blown up and remained unconscious for six days. There was no external wound, but on regaining consciousness he found that he was blind, except that he could just distinguish light from darkness; he was also completely deaf and was unable to speak. He regained his speech in June, after a fortnight's treatment by hypnotism at Plymouth, but his sight and hearing remained unaltered.

When I saw him on September 17th he could only be made to hear by shouting down an ear-trumpet; he kept his eyelids almost closed and constantly twitching with the eyeballs turned upwards. He was extremely depressed, as he had been told by an aurist that he would never regain his hearing, as it was said that nerve deafness of such long duration could not improve, although the drums were intact. He concluded that the blindness would also be permanent. Moreover he was much worried with domestic troubles. It was not easy to hypnotise him, as he was unable to see and all suggestions had to be shouted down his trumpet, but Captain Ormond succeeded at the first attempt. When seen on September 20th he said that his sight was distinctly better, and he was able with difficulty to open his eyes. He was much more cheerful, particularly when we told him that his hearing would also return, as his auditory nerves were no more organically diseased than his optic nerves. On September 30th there was some further improvement, as he could see everything in the outer part of his left field of vision fairly well. In November the patient was given an anæsthetic, and suggestion was tried during the stage of semi-consciousness with marked

success, as on the following day he opened his eyes voluntarily. His hearing gradually improved and by March, 1916, he had recovered completely.

Case 6.—An officer, aged 20, had never used his left eye owing to an extreme degree of hypermetropia. When the other eye was covered he could only see very indistinctly with it, but in spite of this he managed to pass the medical examination on entering the army. He was hit on the left side of the head by the butt end of a rifle in June and was unconscious for a few minutes. When he regained consciousness he at once noticed that he could not see at all with his left eye, although he had hitherto been in the habit of neglecting the blurred image he saw with it. On August 10th he received a slight wound to his left thigh, but continued on duty. The wound had not completely healed and was still somewhat painful when, on August 23rd, he was blown up by a high-explosive shell. On regaining consciousness he found himself being carried on a stretcher. The pain from his old wound drew his attention to his left leg, and he thought he would be unable to walk. When he reached the hospital ship he found that this was the case, although there was no new injury to the leg. He also complained of severe pain above the left eye, which he kept covered by a shade, as he found that the least light greatly increased the headache; when the shade was removed he was unable to open the eye at all. For some time he was in an extremely excited condition, and he slept very badly owing to nightmares. His eye was kept covered by a shade during his journey home from the Dardanelles. On his arrival in England Mr. C. M. Ryley found that beyond the hypermetropia his left eye was normal, although he was quite unable to see anything with it.

He was hypnotised on four occasions. After the first he slept better, the nightmares ceasing and the headache being less severe. On the second occasion, whilst still asleep, the shade was removed from his eye, and he did not discover until half an hour after he woke up that it was no longer present, although up to that time he said that the least light caused extreme discomfort and spasm of the eyelid. After the third treatment he found that he could see almost as well with his left eye as before he was hit on the head. Meanwhile, he was still unable to walk without crutches, although the wound to the leg had completely healed, and there was no physical cause to account for this. When he was hypnotised for the fourth time it was therefore suggested to him that he would be able to walk quite well, and he soon completely recovered.

Disorders of Hearing

Hysterical Deafness. The noise and concussion produced by the explosion of a shell of high power in the near neighbourhood frequently causes deafness. Both ears may be affected, but the one on the side more exposed to the explosion of the shell is generally deafer than the other. The patient is dazed or rendered unconscious by the explosion, and when his mind becomes clear again he discovers that he is quite unable to hear. In most cases the deafness is not due to any peripheral cause, as the drums are intact and Rinne's test is positive (normal). In slight cases the deafness passes off in the course of a few hours, but more frequently it lasts for a few days, and in severer cases it may be absolute and would probably remain permanent if vigorous treatment by suggestion were not carried out. The initial deafness is doubtless due to concussion of the auditory centres and possibly also of the internal ear, but when it persists it is hysterical and due to auto-suggestion, the patient having the fixed idea that his deafness will be permanent. The deafness is rarely accompanied by giddiness and staggering, and the occurrence of nystagmus has not been recorded. Tinnitus is often present; in one case seen with Mr. W. M. Mollison it persisted after the deafness had disappeared. Dundas Grant has noticed that these patients sometimes learn lip-reading with great rapidity,—unquestionable evidence in his opinion of a genuinely high degree of deafness. At the same time, as Grasset has pointed out, the timbre and intonation of the voice remain unaltered, which is very uncommon in severe deafness due to organic disease. According to Milligan and Westmacott a large proportion of these cases have occurred in patients, who have old disease of the ear, or less frequently of the nose or naso-pharynx. This corresponds with the common observation that the effect of a high-explosive shell is generally most marked in any part of the body, the functions of which were more or less abnormal before the occurrence of the explosion. In a small proportion of cases one or both drums are perforated owing to the sudden enormous change in atmospheric pressure; the tear eventually heals with complete restoration of hearing, unless the patient at the moment of regaining consciousness, when he

realises that his hearing is impaired, becomes convinced that he will never hear again and converts by auto-suggestion comparatively slight deafness of one or both ears into complete and bilateral deafness. In many cases the deafness is associated with other symptoms, such as headache, tremor and insomnia, and it is particularly likely to be associated with loss of the power of speech (*vide* p. 22). In a case already described it was associated with hysterical blindness (p. 17). In a few cases, which can only be recognised by the complete failure of treatment, the deafness may perhaps be really incurable and due to hæmorrhage into the cochlea. In a case recorded by James Collier, the patient, who was a very intelligent man, was completely mute and appeared to be deaf. In the course of a written conversation he was asked if he heard anything when spoken to. In reply he wrote: "I can hear your voice quite well, sir, but I cannot gather anything from it." He subsequently recovered completely.

Cases seen at an early stage recover rapidly if they are assured that their condition is not a serious one. On the other hand, if left untreated without any encouragement the patient will become more and more convinced that he is permanently deaf. The worst case I have seen was that of a man who was told by an aurist that his case was hopeless, as he had already been deaf for four months without any improvement occurring. Prolonged treatment by hypnotic suggestion was required to cure him. If he had been told that the kind of deafness from which he suffered never lasted for more than four months and that he would certainly be well in a week, rapid recovery would have resulted.

Hyperacusis.—Hyperacusis is common, the patient being abnormally sensitive to sound and hating the slightest noise; he is unable to sleep in a town, and sudden noises often frighten him. Rapid improvement occurs on being sent to a quiet house in the country, where the silence is not broken by a gramophone or the click of billiard balls. The condition of patients suffering from functional nervous disorders is generally much aggravated by a thunderstorm and still more by a Zeppelin raid; many are reduced to tears and few are able to sleep the following night.

Auditory hallucinations may occur; the patient imagines

he hears shells coming towards him and bursting, or he may hear the whizz of bullets or the blowing of whistles.

Disorders of Speech

(*a*) **Hysterical Dumbness.**—Loss of power of speech is an occasional sequel of the explosion of a shell in the immediate neighbourhood of a patient, and it is sometimes also a sequel of injuries due to other causes, particularly if they are associated with much shock. In some cases there is a history of a blow on the chest or of partial asphyxiation due to burial at the time of an explosion. In addition to being unable to speak aloud, the patient cannot whisper, and in the severer cases he is unable to cough, whistle or make any sound when he laughs. He may be unable to expire with sufficient power to blow out a candle, and in rare cases he cannot put out his tongue. He is generally able to convey his thoughts without any difficulty by writing: he is often in a highly emotional state and in severe cases may be in a constant state of terror, which is prevented from disappearing by frequent nightmares and constantly recurring mental pictures of the horrors he has passed through.

Laryngeal examination shows that the dumbness is distinct from hysterical aphonia, as instead of the isolated paresis of the abductors, characteristic of the latter condition, complete inability to move the vocal cords is present. The dumbness is also quite distinct from aphasia, in which articulate sounds can be made without difficulty and certain words are often reiterated. Moreover the patient understands what he reads and what is said to him, and he can express himself perfectly well in writing; it is clear therefore that the mutism must be due to incapacity of the cortical nerve centres which control phonation and the production of sounds. That this incapacity is functional is proved by the fact that the patient may call out in his dreams, although he is completely mute when awake.

To have one's breath "taken away with surprise" and "to be speechless with rage" express the familiar effects of emotions on respiration and on speech; the excessive shock and the frequently associated emotions of excitement and fear, when a man is blown up by a high-explosive shell in the course of a battle

give rise to similar phenomena but in an exaggerated degree; and the patient on recovering consciousness may become obsessed with the idea that the speechlessness is permanent—the mutism being thus maintained by a process of auto-suggestion.

The dumbness is often associated with deafness, but recovery from the latter or less frequently from the former may rapidly occur, so that the other condition remains alone. Complete mutism often lasts only a few days, after which it may disappear entirely or give place to some form of stammering or less frequently to simple aphonia, when the patient is able to whisper but not to speak aloud. Sooner or later recovery always takes place. It may be spontaneous and occur suddenly or gradually without treatment or as a result of encouragement, but more often speech returns suddenly as a result of direct suggestion, which overcomes the fixed idea of mutism, or of some sudden emotion, which breaks down the subconscious inhibition of speech by surprising it when the patient is off his guard, with his mind for the moment not fixed on his dumbness. Thus speech may return as a result of crying out when in pain or in a moment of excitement, whilst watching a boxing or football match or during a Zeppelin raid, or a mute may suddenly find himself joining in a familiar chorus. It may also return after a nightmare in which the patient calls out, though more often this is not followed by recovery. A patient of Mott's dreamt that he was blown up by a trench mortar and shouted for help. He woke to find himself speaking; he continued to do so, and did not go to sleep again for fear he might lose his speech. I saw a man at Cliveden, who was sleeping when his parents came to visit him, a fortnight after he had become dumb as a result of shell-shock. They woke him up and he at once said "Hullo, mother!" Procter records the case of a soldier who got drunk whilst out of hospital on leave; he "found his voice" and for two days talked and sang without intermission. Sir William Osler tells me that in a case seen by him the patient required an anæsthetic for some trivial operation and speech returned as he recovered from its effect. He therefore advised the administration of ether in two other cases with equally satisfactory results. James Collier, however, found that this method failed in the more severe cases, the patients going under the anæsthetic without making a sound; in these circumstances

the patient may resent the failure and lose confidence in those who are looking after him. If, however, the anæsthesia is accompanied by active suggestion, I do not think that failure will ever occur, and I have found this method succeed in the rare cases in which hypnotism had failed.

An Australian soldier, aged 22, wrote the following letter to his relations on August 21st, 1916 :—

"You may be a little surprised to hear that I am in the Hos. suffering from shell-shock, which has taken away my speech and hearing. It is some sixteen days now since it happened. . . . We were in the trenches and going for dear life, when two of us spotted a German machine gunner in a hole, so we made up our minds to have him. We made a charge at him, and I just remember getting to him when a high-explosive shell burst at my head ; it seemed as if it burst inside my head ; everything went black. I tried to call out and couldn't, and I could not hear my mates—only just a terrible bursting in my head all the time. I never remembered anything more until I came to on the boat. . . . The Drs. have told me that I will get alright in time. . . . I saw a good deal of France. . . . There is not a young man there who is not in the Army. The girls and women work in the fie——"

The letter ended abruptly at this point, as I then entered the ward. The previous day I had hypnotised him without difficulty, but was unable to make any effective suggestions, as the deafness appeared to persist during the hypnotic sleep, so that the suggestions did not reach his brain, and were consequently not acted upon either whilst he remained asleep or after he woke.

He was totally deaf and had heard nothing at all during an exceptionally violent thunderstorm. He was not only unable to speak, but could make no sound of any kind and could not cough.

As no improvement had taken place, he was given ether on this second occasion, after being told in writing that it would have the effect of restoring his speech and hearing. He began to struggle after the first few whiffs, and long before he was anæsthetised he began to repeat the word "Mother," first in a whisper, then louder and louder until he shouted it with a stentorian voice that would have filled the Albert Hall. The etherisation was then discontinued, his limbs never having become relaxed. As he came round, I told him to say various words which he repeated

after me, and I then carried on a continuous conversation with him. When the effects of the anæsthetic finally passed away, he was talking with a normal voice and he had completely recovered his hearing.

His memory, which had previously been unaffected, was now a complete blank from a short time before he was blown up to the moment he had regained consciousness. He had no recollection of having lost his speech or hearing; he was astonished to see the letter he had begun, as he remembered none of the events described in it, and he did not remember having seen me before. In all other respects his memory was perfect.

In this case the patient became speechless from fright at the sound of the explosion, and deaf from the accompanying noise; almost immediately afterwards he lost consciousness as a result of the aerial concussion. The moment he recovered consciousness the fact that he had lost his power of speech and hearing after the explosion recurred to his mind, and as a result of auto-suggestion these disabilities were perpetuated. The subconscious inhibition of speech and hearing was broken down as a result of the loss of control of the higher over the lower cerebral centres when he was under the influence of ether. By keeping these faculties continuously at work whilst the effects of the ether passed off, their recovery was maintained when consciousness returned.

(*b*) **Stammering.**—Stammering may be the primary result of shell-shock, but more frequently it is preceded by complete mutism. In some cases there is a history of stammering when the patient was younger. It is often associated with spasmodic movements of the face whenever an attempt is made to speak, and in one case these spasmodic movements after a time also occurred independently of speech and persisted for a few days after the stammering was cured. In addition to ordinary stammering other speech abnormalities may occur. A sergeant who had been blown up by a high-explosive shell continued to have ghastly nightmares, and remained exceedingly nervous for many weeks. He trembled all over his body whenever anything was said to him, and he could only say three words at a time, each group of three words being invariably preceded by the repetition of "er-er-er" from twelve to fifteen times. This continued

without alteration for many weeks, but slow improvement occurred with hypnotism. Another man had a typical staccato speech, but no other signs of disseminated sclerosis; he had almost complete amnesia and was in a condition of constant terror. An officer, who spoke with extreme slowness and deliberation, but without any actual stammer, rapidly recovered when I sent him to a quiet spot, where in the absence of any audience he recited and made speeches in a loud voice.

The following is typical of several cases in which stammering was cured by hypnotism. Driver D., aged 31, was run over by a loaded transport waggon at Gallipoli in May, 1915. There was no external injury, but his pelvis was fractured. For thee days he was unable to speak at all, although he was perfectly conscious. He then slowly improved, but when he was admitted into the New Zealand War Hospital at the beginning of August, he still spoke with extreme difficulty, and the effort was accompanied by contortions of the whole of his face. Similar contortions of his face also occurred apart from any attempt to speak; they were accompanied by the mental condition characteristic of tics: the patient was able to control them by an effort of will, but he felt miserable when he did so, and was always ultimately forced to give way to the irresistible impulse.

He was readily hypnotised, and it was suggested to him that he would be able to speak without difficulty, and that the contractions would cease. The result was very satisfactory, for as soon as he came round he was able to talk quite normally; the next evening he sang at a concert, and a few days later took part in a play. The facial contortions occurred during the hypnotic sleep and continued afterwards, though less severely; they disappeared after he was hypnotised a second time.

(*c*) **Aphonia.**—Hysterical aphonia may occur in soldiers as a result of exhaustion, and less frequently as a result of the concussion produced by a high-explosive shell, in which case it is generally preceded by complete dumbness of varying duration. It does not differ from the hysterical aphonia seen in civil practice. In a discussion at the Laryngological Section of the Royal Society of Medicine on May 7th, 1915, several speakers referred to the fact that functional aphonia occurs most frequently among

soldiers with some organic disease of the respiratory passages, such as enlarged tonsils, adenoids, antral suppuration, acute laryngitis, deviated septum or phthisis. Hysterical aphonia generally recovers after the application of faradism to the outside or in more severe cases to the interior of the larynx, but if any organic abnormality is present, this of course requires treatment, or a relapse is likely to occur. The patient should be told beforehand that the electricity is certain to cure him; if the treatment is given without any explanation it is likely to do harm by aggravating his emotional condition.

Disorders of Motor Functions

(*a*) **Convulsive Symptoms**: (i) *Hystero-epilepsy.*—Epileptiform convulsions may result from shell-shock. In some cases this is the beginning of true epilepsy (*vide* p. 34), but this is not invariably the case. A New Zealand soldier was rendered unconscious for a few minutes as a result of concussion due to a high-explosive shell. He had had a few fits after falling on his head when eight years old, and the recollection of this probably led by a process of auto-suggestion to the occurrence of convulsions at least once and generally several times a day. I saw him with Captain A. M. Crabtree, who subsequently hypnotised him and suggested recovery, with the result that the fits immediately ceased and have not recurred. The origin may be purely emotional. An officer and his servant were blown up by a shell. The servant did not lose consciousness and ran to fetch a stretcher. On his return the officer, to whom he was greatly attached, made a few convulsive movements and then died. He immediately had a fit and in the following two months he had eleven more. They ceased completely after I had explained their origin and nature to him.

(ii) *Tremor.*—Tremors are very common in men suffering from neurasthenia and shell-shock. I have noticed a slight tremor in officers who have been home on leave for a few days and have regarded themselves as perfectly fit. The symptom tends to be very persistent and is often a source of great worry to the patient, who regards it as an indication of the presence of some serious nervous disease. I have seen the tremor of paralysis

agitans, disseminated sclerosis, and Graves' disease closely simulated. The tremor generally persists when the patient's attention is drawn away from himself, though it is increased when he is excited or spoken to; it always stops during sleep. It may be general or unilateral or may affect both arms or both legs alone.

Irregular trembling of the whole body is not uncommon, especially among men who are in a constant state of terror. It is due to the persistent memory of the horrors which first gave rise to the trembling, and it may continue as a result of auto-suggestion when the patient is no longer terrified. It is always most marked when the patient makes any effort, especially if he is being watched, or when he is asked questions or is excited in any other way. The trembling may be so excessive that it makes walking quite impossible, although the muscular power of the legs may be good. It is often absent when the patient lies down, but begins as soon as he sits up or the moment the feet touch the ground. In the latter case it is due to a kind of false ankle-clonus and gives rise to a curious jerky gait. Improvement is very slow, and hypnotism, which is so useful in most motor disorders of an hysterical nature, often fails, partly owing to the apprehensive state of the patient's mind making it difficult or impossible to persuade him to submit to the treatment, and partly to the condition being for some reason the most resistent to suggestion of all functional disorders.

(b) **Paralysis and Contractures.**—Every variety of hysterical paralysis may occur, either alone or associated with tremor of the affected limbs. In other cases contractures may be present, but they generally involve only a few of the paralysed muscles. Thus a man with hysterical paralysis of his right arm had his fingers tightly clenched, but the rest of the limb was flaccid. In another case a flaccid paraplegia was associated with contracture of the left hand.

The starting-point in most cases of hysterical paralysis is an injury. A man receives a superficial wound of his scalp as a result of a glancing bullet, or as a result of being thrown on to the ground or against a wall or parapet by the explosion of a shell. The brain is not injured and no sign of organic disease is found, but hemiplegia develops—most commonly on the side

of the injury, corresponding with the patient's own idea of the paralysis, which he has heard may result from an injury to the head. In other cases he is injured either directly by a fragment of shell, or indirectly after being blown up by a high-explosive shell or buried under sand-bags. By a process of auto-suggestion the patient develops monoplegia, and in some cases the paralysis spreads and hemiplegia results. Hysterical paraplegia may follow an injury to the back, which is insufficient to cause either a gross organic lesion or even concussion of the spinal cord. The sphincters are unaffected and the plantar reflexes are normal, but the knee-jerks may be exaggerated and false, irregular ankle-clonus, though never true clonus, may be obtained. If there is the slightest bladder disturbance, or if the deep reflexes are lost or the plantar reflexes are extensor, the case is not purely functional.

The gait may be shuffling, the patient being frightened to raise his feet. In other cases he leans forward on two sticks, and is convinced that he cannot move without them. His back often remains bent like that of an old man with spondylitis deformans, even when he stands or lies down, so that he can only rest by lying on his side. I saw an eighteen-year-old soldier with this condition in Lemnos; complete recovery took place as a result of hypnotising him on two consecutive days. Hypnotism on a single occasion cured a similar case in Oxford; the symptoms had developed after the patient had been struck on the back by a falling sand-bag.

Diagnosis of Hysterical from Organic Paralysis.—As hysterical paralysis is a result of auto-suggestion, the paralysis corresponds with the average layman's conception of paralysis. A number of signs occur in organic paralysis, which would not be expected by the uninitiated. They are therefore not observed in hysterical paralysis and serve to differentiate organic from hysterical conditions. In organic hemiplegia involving the face, the platysma of the affected side is paralysed; in hysteria it is unaffected. In organic hemiplegia, if the supinated arm is completely relaxed and then tossed, it falls in a position of pronation, but in hysteria it does not turn. When a patient with organic hemiplegia grips the observer's hand with his normal hand, an associated movement

occurs in the paralysed hand unless the paralysis is too complete, but no such movement occurs in hysteria. The tendon reflexes of the arm and leg are almost always exaggerated on the affected side in organic hemiplegia, though they may be absent or deficient in the early stages, and ankle-clonus is often obtained; in hysteria they are invariably equal on the two sides, and true ankle-clonus is never present. The abdominal reflex on the paralysed side is weak or absent in organic, but equal to the opposite side in hysterical paralysis. The plantar reflex is generally extensor in the former, but invariably flexor in the latter (Babinski's sign). When a patient tries to raise himself from the horizontal to the sitting position with his arms folded and his legs widely separated, the paralysed leg rises higher than the other in organic disease, but remains on the ground in hysterical hemiplegia (combined flexion of the thigh and pelvis—"Babinski's second sign"). In a well-marked case of organic hemiplegia most or all of these signs are present; in hysteria no single one is present. In most cases of hysteria the diagnosis is made still more certain by the gait or some other prominent feature being of a nature quite unlike anything seen in organic disease. Occasionally some inconsistency about the symptoms points to hysteria. An officer became hemiplegic on the right side after being blown up by a high-explosive shell in the Dardanelles. He was taken to Cairo and then sent home. When he said good-bye to his father, who came to see him at Cairo, he shook him strongly by the hand. He told me of this as a remarkable incident when I saw him in London, so it was obvious that he was not malingering. He was cured at once by suggestion during hypnotic sleep.

The following is a good example of the difficulty in diagnosis, when hysterical and organic symptoms are present in the same patient. Private M., aged 29, a champion heavy-weight boxer, was knocked over by the explosion of a high-explosive shell in December, 1914, and remained unconscious for two days. When he regained consciousness, he found that he could not move his right arm or his left leg. Power in both limbs soon returned to some extent, but as soon as he tried to stand, violent involuntary movements occurred in his left leg.

I first saw the patient on April 1st, 1915. His mental condition seemed to be impaired, he only answered questions after

a considerable latent period, and his speech was slow. The whole of his right arm was weak, the grip being particularly feeble. When he clenched his left hand an associated movement occurred in the right hand, but on clenching the right hand no similar movement occurred in the left hand. The muscles of both arms were equally well developed. The tendon reflexes in both arms were brisk, but were no better marked on one side than the other. The patient was unable to localise light tactile stimuli accurately, but otherwise there were no sensory disturbances. All movements of the left leg were somewhat weak. The muscles were equally well developed in both legs. Both knee-jerks were brisk, the left one being slightly brisker than the right. Well-marked ankle-clonus could be obtained on the left side. The plantar reflex was constantly flexor on both sides, but Babinski's second sign was very well marked on the affected side. As soon as the patient attempted to walk, violent involuntary movements were set up in the left leg: the leg moved rapidly from side to side round the point where the toes were in contact with the ground. When a step forward was taken with the right leg, the left leg dragged behind and very irregular movements occurred. The gait seemed to be so obviously hysterical, and the signs pointing to organic disease were so slight, that it was thought that all the symptoms would probably be cured by suggestion. The patient was kept in hospital for a month, but all efforts to cure him by means of suggestion entirely failed. He proved very easily hypnotisable, but even when deeply hypnotised the movements of the leg could not be controlled when he was told to walk. Dr. Batten suggested that etherisation might be of use, but the first whiff of ether had the effect of hypnotising him. In July, 1916, he was in exactly the same condition as when he was first seen, except that his mental condition had greatly improved.

The associated movement of the paralysed hand when the normal hand moves, the slight exaggeration of the left knee-jerk, and the tendency to ankle-clonus, and above all the presence of Babinski's second sign, indicate that some organic changes have occurred in the brain as a result of the concussion. The complete failure of suggestion to produce any improvement raises the question whether all the symptoms, in spite of their unusual character, may not be organic in origin.

In the following case the paralysis was purely hysterical without any organic basis. A Belgian soldier was observing the enemy, when the roof on which he was sitting collapsed, and he fell a short distance into the mud below without hurting himself in any way. It was over an hour before he could extricate himself, as his left leg was embedded in the mud. When at last he was able to crawl away, he found that he could not move his leg, which was fixed rigidly in an extended position. As no improvement occurred during the next few days he was sent to England. When I saw him three months later, his condition was unaltered, and he was still unable to walk without assistance. The left leg was absolutely rigid, but he did not walk as if he had an organic spastic paralysis, but dragged the leg behind him. There was no voluntary movement, and the knee and ankle could only be bent by using considerable force. The whole leg was completely anæsthetic to all forms of stimulation. The plantar reflex of both sides was flexor; the knee and ankle jerks were unobtainable on the left side owing to the impossibility of relaxing the muscles. There was no other evidence of disease in his nervous system. The absence of any organic sign, combined with his extraordinary gait, which was quite different from that of any organic disease, made me quite certain that the paralysis, spasm and anæsthesia were hysterical. Additional proof of this was obtained from Babinski's second sign: when he lay down with his arms folded and legs widely separated and tried to sit up, the normal leg rose in the air and the paralysed leg remained fixed to the ground. The paralysis and rigidity resulted from perpetuation by auto-suggestion of the immobility in an extended position caused by the leg being fixed in the mud. In all probability the anæsthesia was produced by suggestion by one or more of the numerous people, who had examined him during the three months he was incapacitated, as my experience agrees with that of Babinski that hysterical anæsthesia is very rarely the result of auto-suggestion, but is almost invariably produced by the observer. Having decided that the condition resulted from suggestion, it only remained to remove it by suggestion. I passed a strong faradic current through the affected leg and told the patient that this treatment was quite certain to restore his sensation and his power of movement. He improved at once, but was still only able to

walk with difficulty. I therefore hypnotised him, and suggested that he should move his leg in a normal manner. Mr. C. H. L. Harper repeated the treatment on several occasions, and at the end of three weeks he was sufficiently well to be able to return to duty, though he still kept his left leg somewhat stiff when he walked.

I have seen several cases of hysterical paralysis following injury to peripheral nerves. It is very important to recognise this condition, as recovery can then be greatly accelerated. If a nerve is injured in such a way that it becomes functionless, the patient becomes so accustomed to the paralysis that when the nerve recovers sufficiently to convey voluntary impulses, no such impulses originate in the brain. Early in January, 1915, an officer received a bullet wound in his arm, which resulted in complete paralysis and anæsthesia in the distribution of the median nerve. On January 20th Mr. F. J. Steward exposed the nerve and found that it was embedded in connective tissue, but it was not actually divided. The nerve was freed and the wound healed. I saw him on March 9th and found that the anæsthesia and paralysis were still complete, but all the muscles contracted briskly with faradism: this showed that there was no longer any nerve degeneration. With a little persuasion I managed to get him to move his fingers, and by repeating the faradism and persuasion a considerable amount of improvement occurred in the course of a few days. At the end of this time the paresis and partial anæsthesia, which still persisted, were probably organic, the hysterical element having been cured by suggestion and persuasion. In all cases of injury to peripheral nerves, whether they have been completely divided or not, the muscles should be tested from time to time by faradism, and as soon as they respond, the patient should be persuaded to make an effort to contract the muscles voluntarily, as otherwise the paralysis is likely to be maintained by auto-suggestion for several weeks or even months.

Babinski records the case of a patient with paralysis and reaction of degeneration of the muscles supplied by the circumflex, musculo-cutaneous and musculo-spiral nerves caused by a bullet wound in the axilla. Flexion of the hands and fingers was also impossible, although the electrical excitability of the muscles of the front of the arm and forearm three months after the injury was

normal. This showed that the paralysis of the flexors could not be due to an organic nerve lesion; the immediate recovery with purely psychical treatment proved that it was hysterical in origin and not due to nerve concussion. I have observed a similar combination of organic and hysterical paralysis in several cases of bullet wounds involving the brachial flexus.

Pain.—I have seen a number of cases, in which pain, caused by some organic condition, such as an injury to a nerve, has continued with great severity after the removal of the cause. The patient's nervous system was profoundly depressed in every case, and it seems probably that the pain was due to auto-suggestion. In such cases cheerful surroundings and plenty of people to talk to are needed rather than the quiet and isolation which are necessary for neurasthenia.

An officer, from whom Sir Alfred Fripp had removed a bullet in the immediate neighbourhood of the sciatic nerve, still complained of excruciating pain along its course. We discussed the question of injecting the nerve with eucaine or saline solution so as to render it temporarily anæsthetic, but nothing of the kind was required, as he improved directly he was removed from a private room to a large and lively ward. For intractable cases suggestion may prove of value. An officer, who complained of constant severe pain in his back, where he had been bruised by a bullet, which had passed through his coat without penetrating his skin, was rapidly cured by means of the high frequency current, which acted, I expect, as much by suggestion as by any direct analgesic influence.

Diagnosis from Malingering.—The diagnosis of hysteria from malingering is exceedingly difficult, as the symptoms are identical. In hysteria the paralysis or other symptom is produced by auto-suggestion and corresponds with the patient's own conception of paralysis; it is consequently indistinguishable from the paralysis which the malingerer voluntarily assumes. Though malingering may be suspected in other cases, it can only be diagnosed with certainty under two conditions. Occasionally an unskilful malingerer may be detected *flagrante delicto:* the appropriate treatment for a paraplegic man, who is discovered walking in the ward when he

thinks he is alone and unseen, is to send him to the military authorities for punishment. Very rarely a malingerer confesses that he is shamming: he should be sent back to duty at once, but without punishment. Pure malingering is very rare in the British and French Armies. Conscious exaggeration of symptoms and conscious prolongation of incapacity, which is primarily involuntary, are comparatively common. Malingering may in this way be associated with both organic disease and hysteria. It should also be remembered, as Gilbert Ballet has pointed out, that malingering may end in hysteria; a man who pretends to be paralysed for a sufficiently long period may end by genuinely believing he is paralysed, just as the German people have repeated the official lies as to the cause of the war so frequently that many now doubtless believe in the truth of what they originally knew was untrue.

The War and Organic Nervous Disease.—There is no doubt that organic nervous diseases are aggravated by factors, such as fatigue and worry, which are often erroneously supposed to lead only to functional disorders, and in years to come we shall probably see cases of paralysis agitans, disseminated sclerosis, and other organic nervous diseases, in which the war will prove to have been the exciting cause. Bruce has recorded the case of a soldier, who had had symptoms of disseminated sclerosis twelve years before, but then had a complete remission; the symptoms returned suddenly as a result of severe wetting during a long route march. A colonel, who went out with the original expeditionary force, regarded himself as perfectly fit, though he had occasionally suffered from pains in the legs, which had been ascribed to rheumatism and neuritis. He had an exhausting time during the great retreat, and was invalided home directly afterwards. I found that he was suffering from typical and severe tabes, which had doubtless been present in a latent state before, but had become acute as a result of over-fatigue. I have not yet seen any cases of general paralysis of the insane, but the mental strain and physical fatigue of active service would be quite enough to give rise to the disease in an individual predisposed by syphilis, for what leads to neurasthenia in a normal man is likely to cause general paralysis in a syphilitic; such a case has indeed been recorded by Mott.

Fearnsides has observed that patients with shell-shock, in whom the Wassermann reaction is positive, improve with remarkable rapidity with anti-syphilitic treatment, although other treatment may have completely failed.

Among the numerous cases of sciatica I have seen, a large proportion occurred in soldiers who had suffered from it before the war, the return of symptoms being due to exposure and fatigue. Exaggeration and voluntary prolongation of incapacity are particularly common in men who have had sciatica.

Epilepsy.—True epilepsy sometimes develops as a result of shell-shock, but Mott has shown that in fully four-fifths of the cases the affected individuals were predisposed by having previously suffered from some form of major or minor epilepsy or by having an epileptic parent. In some of the remaining cases the shell-shock was accompanied or preceded by an actual injury to the head. The possibility of hysteria should always be considered, even if there is a history of former fits, unless perfectly typical epileptic convulsions are seen, or the patient micturates, defæcates, or bites his tongue during the fit.

Treatment

Complete physical and mental rest are essential in the treatment of neurasthenia and shell-shock. The patient must be kept in bed until he no longer feels tired. In very slight cases a week or fortnight may be enough, but two or three months may be required, especially in traumatic cases. No visitors should be allowed, except one or two relations or friends whom the patient particularly wishes to see, and frequent changes of nurses are most undesirable. Severe cases should be isolated until considerable improvement has occurred. Quiet is essential, and recovery is most rapid if the patient is kept in the open air, but protected from bright sunshine. The patient should not read at first, but he may be read to, and all exciting literature should be avoided. He should not talk about his experiences in the war, but should try to forget the scenes he has passed through. Cheerful companionship is most helpful, and when some improvement has occurred every effort should be made to keep the patient amused and interested in subjects which help to divert his mind

from the horrors he has witnessed. Men should be sent to convalescent hospitals in the country as soon as the severer symptoms, and especially the nightmares, have disappeared, but they should not be left to themselves, but given definite prescriptions for work in the garden, farm, or carpenter's shop, beginning with half an hour morning and afternoon and gradually increasing till they can do a full day's work, by which time they are fit to return to duty.

Treatment by Suggestion and Hypnotism.—Hysterical symptoms in soldiers have proved very amenable to treatment. Some cases sooner or later recover spontaneously, but others show no tendency to improve if left to themselves. Simple persuasion and encouragement are often all that is required, especially in early cases ; but many are very resistant, especially if the condition has already lasted for more than two or three weeks. On the other hand, suggestion, especially with the aid of hypnotism, rarely fails. If simple persuasion were equally successful, it would doubtless be preferable to hypnotism, but the latter produces more rapid results, two or three days being generally sufficient to complete the cure in early cases, and hypnotism is often the only treatment which has any chance of being successful in late cases. But even with hypnotism the rapidity in which recovery occurs varies inversely with the duration of the symptoms. Consequently if simple persuasion and encouragement do not lead to definite improvement in the course of a few days, hypnotism should be employed without delay, and in cases of any severity, especially if they are first seen after the symptoms have been present for some time, or if other methods of treatment have already been tried without success, the patient should be hypnotised without waiting to try the effect of other measures.

If the patient is amenable, hypnotism very rarely fails. It is impossible to hypnotise men who are in a constant state of terror, as they resist every attempt and become even more terrified than before, if the hypnotist perseveres in his efforts. I found it quite impossible to hypnotise three such patients whom I saw in Oxford with Lt.-Col. W. Collier, as on simply entering the rooms in which they were isolated, they shook from head to foot, and showed every sign of extreme fear before a word had been said. A year later one had completely recovered, another was very

much better, and the third, a man of 56, was almost as bad as ever; no treatment beyond rest and encouragement had been employed. In a recently published paper on functional nervous affections in soldiers two cases of hysterical paraplegia and one of hysterical monoplegia in Belgians are described: re-education, persuasion, strong faradisation, etherisation and isolation were tried in vain, although the patients gladly submitted to every form of treatment. I feel convinced from what I have seen of the effect of hypnotism on Belgian as well as British soldiers that it would have been successful in these cases, especially if it had been tried before the faradisation, which is only a somewhat rough, though often useful, method of applying suggestion.

The objection is sometimes raised that hypnotism treats symptoms without dealing with the underlying abnormal condition of the nervous system. This may be true in non-traumatic cases of hysteria seen in civil life, but it does not apply to the hysterical manifestations which occur in soldiers. The symptoms have generally been produced by some quite exceptional circumstance, such as the explosion of a big shell, in spite of the fact that the individual's nervous system was either normal or somewhat exhausted as a result of the strain and stress of war; in any case it was in a very different condition to the quite abnormal nervous system of the young woman, who in civil life is particularly liable to hysterical symptoms, and for whom other methods than hypnotism are almost always successful and are certainly preferable.

With the disappearance of his hysterical symptoms a soldier may often be regarded as cured and fit again for active service. More frequently, however, other symptoms are still present, especialy if he is suffering from shell-shock, but the disappearance of the most obvious and distressing symptom, such as blindness, deaf-mutism, paralysis, insomnia and nightmares, removes his chief source of worry and encourages him so greatly that the other symptoms indirectly benefit to a remarkable degree. It is easy to understand how the return of vision to a man, who is suffering from both hysterical amblyopia and neurasthenia, will do his neurasthenic symptoms more good than months of rest, encouragement or other treatment.

Everybody who has to deal with soldiers suffering from neuroses of any kind ought to learn to hypnotise them himself. There

is no difficulty about it, as shown by the fact that every one of the numerous clinical clerks in my Neurological Department at Guy's Hospital who has tried has succeeded after the first or second attempt, and the process is very much less fatiguing both to the patient and the hypnotist than such methods of treatment as persuasion and re-education.

It is essential for success that the hypnotist should feel convinced that the patient's symptoms are not organic or are at most only in part organic. It is sometimes impossible to distinguish with certainty between hysteria and malingering; but the distinction is of no great importance, if, when malingering seems possible, Babinski's suggestion is followed, and the statement is made in the man's hearing that "nervous" cases are cured by the treatment to be adopted, but that "skrimshankers" are not. If malingering is not merely possible but probable, the same statement should be made, but a painful faradic stimulation of the affected part should be substituted for hypnotism.

Whatever treatment is adopted, the encouragement produced by the presence of cured patients in the same ward and their exhibition to those about to be treated, is most helpful.

It is quite unnecessary to use any elaborate methods, as the majority of patients are so anxious to get well that they are very easily hypnotised. The patient should be alone in a room or surrounded by screens, and lie comfortably with his muscles relaxed in the position he would assume if he were about to go to sleep. He should be told that the treatment is certain to cure him rapidly, and examples of similar cases may be related to him. He is instructed to think of something pleasant and not to listen to what is said to him: he may for example try to picture his home to himself and imagine himself walking through the various rooms. He may fix his eyes for a few moments on a lens or other object before closing his eyes, but this is quite unnecessary, as shown by the ease with which patients with hysterical amblyopia and blepharospasm, who cannot open their eyes, can be hypnotised. One hand can be put firmly on the forehead and the other may stroke the eyes, but this also is not essential. The patient is next told in a low monotonous voice that he is going to sleep, that he is getting drowsy, that he will soon be asleep, and so on for two or three minutes. Curative

suggestions are then made without attempting to ascertain whether he is really hypnotised, as the success of the treatment does not depend upon the degree of unconsciousness, and it is even unnecessary for consciousness to be lost at all. A blind patient is told that his eyes are not really diseased, but that his sight is only temporarily lost, that he will be able to see again when he wakes as well as he ever could; a paralysed man is told that his loss of power has disappeared although he has up to now been unable to realise it, that he will move his limbs perfectly as soon as he wakes and that the weakness will never return. Suggestions of this sort are repeated over and over again, and after five or ten minutes the patient is told to wake. He should not as a rule be examined at once, but should be allowed gradually to realise for himself how much better he is. In some cases a single treatment is sufficient, but more often it must be repeated once or twice, and occasionally several times. It is often possible to make a paralysed man move the affected limbs whilst he is asleep: if this is done it is a good plan to let him wake whilst he is still performing the movement. On three occasions I have allowed paraplegic patients to wake and find themselves walking quite normally. One man whose fingers were so tightly clenched that it was impossible to force them open, the attempt producing considerable pain, was hypnotised and told to open his hand: he did this, and the moment he woke he looked with astonishment at the palm of his hand, which he had not seen since the contracture developed after he had been blown up by a shell four months before.

Drugs.—Recovery is often greatly accelerated if a small dose of bromide, such as gr. v, is given two or three times a day. Opium must be avoided, as it often aggravates the symptoms and increases the excitability and restlessness of the patient. No alcohol should be allowed. It is very badly tolerated by all patients who have had shell-shock, a quantity which formerly produced no ill-effect being often sufficient to intoxicate. In some cases of insomnia the effect of suggestion is greatly increased by the use of drugs, and when there are no dreams the latter may be used alone. I have found sodium diethyl-barbiturate (medinal) gr. xv with acetyl salicylic acid gr. xv the best combination. The dose of the former should be reduced by gr. i every night until only the latter is given, and this can then be gradually given up.

References

W. Aldren Turner: *Lancet*, 1915, I. 833.

C. S. Myers: *Lancet*, 1915, I. 316; 1916, i. 60 and 608, and ii. 461.

F. W. Mott: *Lancet*, 1916, I. 331, 441, and 545.

Discussion on Shell-Shock without Visible Signs of Injury: *Proc. Roy. Soc. Med.*, Sections of Psychiatry and Neurology, ix. 1916.

G. Elliott Smith: *Lancet*, 1916, I. 813.

H. Wiltshire: *Lancet*, 1916, I. 1207.

Review of Neurology and Psychiatry, 1916: Special Numbers on "Neurology of the War."

Revue neurologique; Special Numbers on "Neurologie de Guerre," mai-juin et nov.-déc., 1915; avril-mai, 1916.

Ravant: *Presse médicale*, 1915, p. 313.

Feiling: *Proc. Roy. Soc. Med.*, Neurological Section, VIII. 67, 1915.

A. P. Procter: *Lancet*, 1915, II. 977.

T. E. Harwood: *Brit. Med. Journ.*, 1916, I. 551.

A. F. Hurst and A. W. Ormond: *Lancet*, Jan. 1st, 1916.

A. N. Bruce: *Review of Neurology and Psychiatry*, XIV. 1, 1916.

W. Milligan and Westmacott: *Proc. Roy. Soc. Med.*, Laryng. Section, VIII. 114, 1915.

Zeitschrift für die gesamte Neurologie u. Pathologie; Special Numbers on "Kriegsneurosen," XI. 321, 1915; XII. 1, 1915; and XII. 317, 1916.

Neurologisches Centralblatt, XXXV. 225, 1916

CHAPTER II

DYSENTERY

Dysentery is the generic name originally applied to cases of colitis, in which frequent stools containing blood and mucus were passed and tenesmus was present. The discovery of the *Entamœba histolytica* and the more recent discovery of the *Bacillus dysenteriæ* resulted in the differentiation of amœbic and bacillary dysentery, which are now known to be distinct in their geographical distribution, morbid anatomy, and to some extent in their symptoms, and to require distinct treatment. The modern tendency has been to restrict the term dysentery to infective colitis caused by these organisms, even if no blood and mucus are passed and no tenesmus is produced. It is, however, more logical to include all forms of infective colitis in which blood and mucus may be passed and tenesmus may occur, as the disease produced by certain other protozoa closely resembles amœbic dysentery, and there is a greater similarity between the morbid anatomy of the ulcerative colitis, which occurs sporadically in England, and chronic bacillary dysentery than between bacillary and amœbic dysentery.

(i) Amœbic Dysentery

Amœbic dysentery is common in tropical and sub-tropical countries, but it had never occurred in epidemic form in Europe until the summer of 1915, when it caused a large amount of invaliding at Gallipoli among the British troops; they had probably brought it with them from Egypt, as the disease never became common among the French troops at Sedd-el-Bahr.

Parasitic amœbæ—entamœbæ—are distinct from the non-parasitic amœbæ of the soil and water in their morphological characters. They have not yet been cultivated artificially, and

they become parasites in the intestine when given by mouth, whereas the non-parasitic amœbæ can be cultivated, but spontaneously disappear from the fæces within a week of being swallowed.

There are two varieties of entamœbæ, which are structurally distinct; one—the *Entamœba coli*—is a harmless parasite, found in the stools of many normal individuals, and the other—*Entamœba histolytica* (or *E. dysenteriæ*)—is the cause of amœbic dysentery. The *Entamœba coli* produces no lesions when given to kittens by mouth, but the *Entamœba histolytica* produces characteristic ulcers in the colon and abscesses in the liver, which contain the pathogenic amœbæ but no bacteria.

Different forms of the entamœba of dysentery have been described. Wenyon has now definitely proved that the large form (15 to 30μ in diameter), which invades the tissues, and the *E. minuta* (10 to 20μ), which lives like the *E. coli* on the surface of the mucous membrane, are really two forms of a single organism, the *E. histolytica*. The large form is the only one found in the stools of acute dysentery; its size is about the same as that of *E. coli*, but it is more refractile and greenish, with a clearer distinction between ectoplasm and endoplasm, its pseudopodia being formed of highly refractile ectoplasm alone, its nucleus is smaller and less easy to detect, and its movements are more active; it may contain many red blood corpuscles, though none are found in *E. coli*. When the acute symptoms abate the *E. minuta* replaces the large form. Both forms reproduce by simple division, but some of the former cease to multiply, become spherical and encyst themselves by secreting a transparent shell. The cyst is completely passive and is excreted in the fæces. Its single round nucleus divides into two and then into four. The four nuclei, on account of which it has been described as the *E. tetragena*, distinguish it from the cyst of *E. coli*, which contains eight nuclei; the latter is also larger (15 to 20μ instead of 10 to 14μ) and less refractile. The difference between the cysts is of great importance, as the active stage of the minuta form of *E. histolytica* closely resembles that of *E. coli*.

The active forms of *E. histolytica* die too quickly outside the body to take any great part in spreading the disease. Amœbic cysts survive many days in moist fæces and in water; the

infection can therefore be carried in particles of fæces, which are moist inside though dry outside, and pieces of paper or leaves contaminated with moist fæces, and in contaminated drinking water. As the cysts are rapidly killed by drying, they cannot be carried in dust.

Wenyon's investigations have shown that flies are much the most important means of spreading the disease. They ingest fæces, which they begin to excrete within half an hour, and in twenty-four hours the excretion is almost complete. If the fæces contain amœbic cysts, the latter can be found during the whole of this period in the intestines of the fly. As a fly generally excretes whenever it ingests food, it deposits fæces containing amœbic cysts on any human food upon which it settles, if it has had access to infective fæces in the preceding twenty-four hours. A considerable number of house-flies caught by Wenyon in different parts of Alexandria, including one caught in a cookhouse, deposited fæces containing cysts of *E. histolytica* and *E. coli*. The infection is probably rarely if ever carried by the fly on its legs, body, or proboscis, as it cleans itself after leaving the fæces, and the traces still left dry so rapidly that no amœbic cysts could survive long enough to be deposited on food.

The stools of convalescent and contact carriers are a more fertile source of infection than those of patients already suffering from dysentery, as the encysted amœba is present in largest quantities in the formed stools of patients who have recovered from dysentery—convalescent carriers—and of individuals who have never had definite dysenteric symptoms, the disease having been latent or the submucous tissue having entirely escaped invasion,—contact carriers. Fatal dysentery can be produced in cats by feeding them on the cyst-containing fæces of carriers, who have no symptoms of dysentery. Owing to the extreme frequency of defæcation, soiling of the patient's clothes and skin is common, and hospital attendants may infect themselves, if they are careless about washing their hands. Defective personal hygiene is a frequent cause of the spread of the disease in camps, as the hands are likely to be soiled with infective material in the latrines, and if they are not thoroughly washed, the food becomes contaminated.

The cyst wall is unaffected by the gastric juice, but is

digested by the pancreatic juice. Each of its four nuclei develops into an active amœba, which burrows from the lumen of the colon through the mucous membrane into the submucous tissue.

Morbid Anatomy.—The essential lesion in amœbic dysentery is an inflammatory round-celled infiltration of the submucous tissue of the colon, caused by the invasion of the amœbæ. The proximal part of the colon is first involved, and even when the whole colon later becomes affected, the disease is generally most advanced in the cæcum. The appendix is occasionally affected, but the ileum is always spared.

Small raised red dots first appear as a result of congestion of the mucous membrane over an area of submucous infiltration; the centre of the congested area soon becomes necrotic and yellow, and the necrotic tissue finally disappears, leaving a spreading ulcer with an overhanging margin and the infiltrated submucous tissue as its base, or a flask-shaped cavity containing pus. The affected areas are greatly thickened and are raised above the adjacent mucous membrane, which remains comparatively healthy; even in the severest cases areas of normal mucous membrane remain, especially in the distal half of the colon. In the worst cases black sloughs of necrosed mucous membrane, which may be several inches in diameter, separate, and are passed in the stools, or are found after death attached here and there to the edge of a large ulcer. In these cases the amœbæ pass between the muscle fibres to the subserous coat, and the round-celled infiltration extends to the muscular coat and peritoneum, which is covered with purulent fibrinous patches.

In very chronic cases and in the process of healing the ulcers become slightly depressed, round or oval in shape; they finally leave dark rounded scars.

The amœbæ may invade the small submucous veins which carry them to the liver, where they give rise to inflammation and suppuration (*vide* Chapter III). In rare cases abscesses also form in the brain and spleen. I saw two cases in Salonica, in which all the symptoms of cerebral abscess were present; both patients had had dysentery at Gallipoli, and rapid and complete recovery followed treatment by means of emetine injections. It seems probable that they were suffering from amœbic

encephalitis, corresponding with the hepatitis which precedes the actual formation of an abscess in the liver.

Symptoms.—The incubation period is short. Crean records the case of a soldier, who landed in Alexandria from England at 4.30 p.m. on a Friday. He had never been abroad before. He at once marched to Mustapha, where there were several cases of amœbic dysentery. At 9 a.m. on Sunday—forty hours after his arrival—he passed blood and mucus, which contained amœbæ.

The onset of amœbic dysentery may be acute, but in a considerable proportion of cases the disease begins as chronic diarrhœa or as diarrhœa alternating with constipation. In acute cases ten to twenty or even more stools are passed a day. When the disease has fully developed, blood and mucus are almost always present in the stools, which consist of a few drachms of greenish yellow or dirty brown mucus, or a grey muco-purulent mass, suspended in semi-fæculent or serous fluid, which is often blood-stained; the blood is either intimately mixed with the stool or is present in streaks. In other cases large separate fragments of mucus stained bright red are seen. The presence of pus, infiltrated sloughs and black sloughs indicate that gangrene is present. At least one stool should be inspected every day. Additional information can often be obtained by diluting it with water and allowing it to stand a couple of minutes, when the mucus and sloughs sink to the bottom; the fluid is then poured off with most of the fæces, and the residue is washed repeatedly until no fæcal matter is left.

If a microscopical examination is made within half an hour of the passage of a stool, active amœbæ can generally be found without difficulty in the blood-stained mucus, but they may also be present in mucus without obvious blood and in watery stools free from blood and mucus. The amœbæ are generally scattered among numerous pus cells and often numerous red corpuscles with some epithelial cells.

Griping pains, which are worst shortly before and during defæcation, are always present to a greater or less extent across the lower part of the abdomen. Tenesmus, the painful bearing down and straining sensation, which occurs during and for some

time after defæcation, is common, but it is only severe if the rectum is ulcerated.

The thickened colon can often be felt, especially in the right and to a less extent in the left iliac fossa, as a very tender tumour; when the cæcum is chiefly involved, appendicitis may be simulated.

Constitutional symptoms are generally slight and, apart from complications, fever is either completely absent or low and intermittent, even in acute cases. Leucocytosis is generally present, and there is a relative increase in the mononuclear cells; in severe cases the number of leucocytes per c.mm. often exceeds 20,000 and sometimess even 30,000.

In the fulminating gangrenous type of amœbic dysentery the patient passes very numerous stools, which often consist of blood with a little mucus but no fæces. When extensive gangrene occurs, the colon may become paralysed, and only a few stools containing black sloughs are passed. Abdominal pain is severe, and palpation shows that parts of the colon are greatly thickened and extremely tender. In such cases perforation and general or local peritonitis may occur.

Mild and unrecognised amœbic dysentery is a not uncommon precursor of hepatic abscess; a history of slight diarrhœa without the passage of blood, often alternating with constipation and sometimes accompanied by pain in the right side of the abdomen, is obtained. The comparatively mild symptoms are probably due to the lesion being confined to the cæcum and ascending colon, which may be tender and slightly thickened. In such cases amœbæ are difficult to find in the stools, unless frequent search is made for a little blood-stained mucus. The diarrhœa, which was common among the troops at Gallipoli, was probably in many instances a mild form of amœbic dysentery. The disease may even be completely latent, ulceration being found post-mortem in patients dying from amœbic abscess of the liver or from some intercurrent disease.

In chronic amœbic dysentery, which should never occur with efficient treatment, symptoms may continue for months or years, often with long intermissions; emaciation, anæmia, and general asthenia result, and the patient ultimately dies from exhaustion or hepatic complications.

Several cases of sciatic neuritis and a smaller number of neuritis affecting other nerves were observed in association with the outbreak of amœbic dysentery in the Mediterranean Forces.

Diagnosis.—In any neighbourhood in which dysentery is known to occur, every case of diarrhœa should be regarded as a possible case of dysentery, and if blood or mucus or both are passed or if tenesmus is well marked, it should be regarded as a probable case of dysentery. Other causes of similar symptoms, the most important of which is a growth of the pelvic colon or rectum, should not be forgotten; I found an inoperable growth in the rectum of a governess sent home from Persia for what was supposed to be chronic dysentery, and I saw a case in Lemnos, in which a man with a similar growth had been treated for some weeks with emetine. A careful inquiry into the history and in doubtful cases a rectal examination should prevent such a mistake being made.

If symptoms of appendicitis occur in a patient, in whom there is a possibility that amœbic dysentery is present, the stools should be examined for amœbæ, and the effect of emetine injections should be tried before advising operation, except in severe cases in which the delay caused by failure of the treatment might prove dangerous.

The clinical differentiation of amœbic and bacillary dysentery will be discussed after the symptoms of the latter have been described. A definite diagnosis can only be made by the discovery of the *Entamœba histolytica* in the stools. Various features have already been mentioned which distinguish the *Entamœba coli* from the *Entamœba histolytica* both in the active and encysted states, but whenever active amœbæ containing red corpuscles are found in the blood-stained mucus passed by a patient with dysenteric symptoms, the diagnosis of amœbic dysentery can be safely made.

In extensive epidemics in war time, when it is impossible to examine the stools of every patient microscopically, treatment with emetine should at once be instituted in cases arising in a unit, in which the disease is known to have occurred; rapid improvement can be regarded as almost conclusive proof of the amœbic origin of the dysentery.

Prognosis.—If emetine is promptly given death should hardly ever occur. When treatment is delayed the prognosis is less good, and the actual dysentery is likely to be followed by chronic colitis, which may persist for many months. Incontinence of fæces, persistent vomiting, hiccup and the passage of sloughs are the most serious symptoms.

Prophylaxis.—Neither chlorination nor the addition of acid sodium sulphate tablets to water destroys encysted amœbæ. All drinking water should therefore be boiled when amœbic dysentery is prevalent, and no raw vegetables in the form of salads, and no raw fruit unless it is carefully peeled should be eaten. In severe epidemics a tablet containing gr. 1/6 of emetine hydrochloride, which does not cause any digestive disturbance in the majority of individuals, or a smaller dose if this cannot be tolerated, should be taken in food or water before meals in order to destroy any amœbæ which may be swallowed, before they have time to penetrate the mucous membrane of the colon. Every case of diarrhœa lasting for more than twenty-four hours should be regarded as possibly due to dysentery and treated in the manner described in the note at the end of this chapter. A patient who has had amœbic dysentery should not be allowed to return to his unit, until he can take a full diet without getting diarrhœa and his fæces have contained no blood or mucus for a fortnight. He should continue to receive subcutaneous injections of emetine after his apparent recovery, as described in the section on treatment. The fæces of all troops should be passed into dust- and fly-proof receptacles containing cresol, and whenever possible they should be incinerated; failing this they should be deeply buried. However thoroughly this is done, flies will continue to carry infection from the fæces of native carriers. Efficient fly-traps should therefore be kept at the entrance to all cook-houses and messes, both of which should, whenever possible, be protected by fly-proof netting. As in spite of these precautions flies are certain to enter, all food should be kept covered. Special attention should be paid to the health of cooks, and no man should be allowed to have anything to do with the preparation of food if he is suffering from diarrhœa. Before leaving their wards medical officers, nurses, and orderlies, who are looking

after dysentery cases, should wash their hands with cresol, which Wenyon has shown is the best antiseptic for destroying amœbic cysts.

Treatment.—The patient should be kept warm in bed until the diarrhœa ceases. The diet required for all forms of dysentery is the same. It should be entirely fluid at first, and in very acute cases nothing but barley water, albumin water or whey should be allowed for twenty-four hours. With this exception it should consist in the early stages chiefly of milk, citrated by the addition of two grains of sodium citrate to each ounce. Milk soured by a pure culture of lactic acid bacilli, yoghurt or the similar Egyptian preparation called laban zebady is very useful when available, as it renders the conditions in the colon unfavourable for the development of dysenteric and putrefactive organisms. Arrowroot and cornflour made with water and sweetened can be given from the onset; when improvement occurs it can be made with milk, and custard, thick purées of potatoes, peas or lentils, and milk puddings may also be given. Meat extracts should be avoided. When the stools begin to be formed the diet can be gradually increased.

Directly the diagnosis has been made by the discovery of the *Entamœba histolytica* in the stools, gr. i of emetine hydrochloride should be injected subcutaneously, and gr. i injected once, or gr. ½ injected twice, daily for the next twelve days. In slight cases treatment should be equally prolonged; in very severe cases gr. ½ to i dissolved in 5 c.c. of normal saline solution should be injected intravenously, and in somewhat less severe cases gr. i can be given subcutaneously twice a day for as long as may be necessary, but there should be an interval of a few days after each period of twelve days. If it is impossible for a prompt pathological examination of the stools to be made, the treatment should still be instituted without delay; if no improvement occurs, it may be safely assumed that the diagnosis of amœbic dysentery is incorrect. In the rare cases of amœbic dysentery, in which emetine fails more or less completely, a mixed infection is generally present, and antidysenteric serum should be given in addition to the emetine. It is unnecessary to persevere with emetine until the stools are solid, as after the blood and mucus

have disappeared the stools may remain loose for a time owing to the post-dysenteric and non-specific colitis still present. They may rapidly become solid on discontinuing the emetine, which apparently acts as a mild intestinal irritant. If any blood or mucus reappears in the stools, gr. i of emetine hydrochloride should be injected every night for six nights; it is unusual for more than one such relapse to occur. In the very exceptional cases in which emetine injections cause vomiting, this may be prevented by the simultaneous injection of a small dose of morphia.

Amœbic cysts are not killed by 1 in 100 emetine after nine hours, whereas active amœbæ are rapidly killed with a strength of 1 in 100,000, and the former are often still present in the lumen of the colon of patients who have apparently recovered from an attack of dysentery. Active amœbæ may develop from the cysts and cause a recurrence of symptoms if the patient's general health is depressed or his colon is irritated, and a chronic infection of this sort may give rise to amœbic hepatitis or abscess of the liver. So long as amœbic cysts are still present in the bowel the individual remains a source of danger to others. For these reasons an injection of emetine-bismuth-iodide (gr. i) should be given thrice daily for fourteen days to all patients who have had amœbic dysentery. By these means amœbæ, which have developed from the cysts since the last injection, are destroyed; one or two drachms of sodium sulphate should be given at the same time in order to clear away as many of the cysts as possible. Rectal injections of quinine bihydrochloride (gr. ½ to the ounce), which exerts a powerful destructive action on amœbæ, may be given occasionally, if the response to emetine treatment is slow, but not in the acute stages when there is danger of perforation.

When pain is excessive and especially if it causes insomnia, and when very severe diarrhœa persists in spite of treatment and is leading to exhaustion, which would probably be fatal if it continued, a hypodermic injection of a quarter to half a grain of morphia should be given and repeated, if required, every four hours, with the addition in the former case of a hundredth of a grain of atropine. I have found charcoal very effective in diminishing the frequency and deodorising offensive watery stools: at the same time flatulence, which is often excessive and is an important cause of colicy pain, is greatly reduced. Half an

ounce of charcoal to a teacupful of sweetened arrowroot makes a palatable food and should be given three times a day in the more acute stages, and a single dose should be given last thing at night as long as the stools remain soft.

Hot hip baths are useful for tenesmus; when the latter is severe and persistent, the individual ulcers should be swabbed with a saturated solution of silver nitrate through a proctoscope.

If excessive diarrhœa results in collapse, three or four pints of hypertonic saline solution, as recommended by Rogers for cholera, should be injected intravenously: two drachms of sodium chloride are dissolved in each pint of water. In less urgent cases, normal saline solution (gr. 90 to the pint) may be injected subcutaneously. No drug, with the possible exception of pituitary extract, is of the least value in such cases; the subcutaneous injections of adrenalin chloride (1 in 1000), which has sometimes been recommended, is worse than useless, as even in healthy individuals, three minims often cause tachycardia, and a larger dose produces a feeling of collapse and impending death.

Since the treatment has become so much more effective as a result of using emetine subcutaneously, neither appendicostomy nor any other operation ought ever to be necessary.

Convalescent patients are very liable to have attacks of diarrhœa on exposure to cold and wet, or if care is not taken with their diet, as the colon remains irritable for many months, even after the infection has been completely cured by means of emetine. Except in mild cases of very short duration, it is advisable for the patient to live for some months afterwards in a warm and equable climate in a country where the disease is not endemic, and he should wear a cholera belt; he should take no green vegetables, except as purées, no salads or pickles, and no fruit skins or pips, whether raw, cooked or in jam, for at least two months after the symptoms have disappeared. The tendency for constipation to develop should be overcome by the use of liquid paraffin or sodium sulphate, and not by vegetable aperients, which are likely to cause intractable diarrhœa. Attention should be paid to the teeth in order to prevent secondary infection of the bowel. There is no evidence, however, that the amœba found in many cases of pyorrhœa alveolaris has any connection with dysentery, though morphologically it resembles the *E. histolytica*.

(ii) Bacillary Dysentery

From what is now known about its geographical distribution it is highly probable that the dysentery which has always been common in armies during war-time was bacillary and not amœbic. Out of the 30,000 English soldiers who fought in the Crimea, 7883 suffered from dysentery, and of these 2143 died; in the South African War there were 38,108 cases with 1342 deaths. In the Federal army during the American Civil War there were 285,000 cases, as many as 21 per thousand troops being attacked in 1864.

Bacillary dysentery was the more common form of the disease in the British Mediterranean Forces until June, 1915; during July, August, and September a large majority of cases were amœbic, but with the colder weather after October very few additional cases developed, and bacillary dysentery became once more the prevailing type, though its frequency gradually diminished, until at the end of the year there was very little dysentery of either kind. Bacillary dysentery was common, but amœbic dysentery rare in the summer of 1916 at Salonika; it also occurred among all the armies fighting on the Western front.

The bacillus of dysentery was discovered by Shiga during an epidemic in Japan in 1898; varieties of the organism, differing in their cultural properties, were isolated by Flexner and by Strong in 1900, and by Hiss and Russell—the Y-bacillus, which is very similar to Flexner's—in 1903. The bacillary dysentery in the Mediterranean Forces was chiefly caused by Shiga's bacillus, and that in France by the Y-bacillus. In some cases bacilli have been found which differ slightly from all previously isolated varieties.

The disease is spread in exactly the same way as amœbic dysentery, and the *B. dysenteriæ* has been found in the excreta of flies up to three days after they had fed on infected stools. The stools of convalescent patients may still contain the bacillus for as long as a year after infection. So-called "contact carriers" have generally had a mild diarrhœa, the nature of which has not been recognised, as there is no doubt that the *B. dysenteriæ* can cause slight catarrhal colitis, in which loose stools containing no blood and little or no mucus are passed.

Morbid Anatomy.—In acute bacillary dysentery all or nearly all of the colon and the last one to three feet of the ileum are involved. In chronic cases the changes are generally confined to the lower half of the colon.

The disease begins as an acute inflammation of the whole mucous membrane with areas of coagulation necrosis, which later give rise to superficial ulcers, the submucous tissue and the muscular and peritoneal coats escaping more or less completely. In a typical specimen dark greenish grey necrotic areas and irregular superficial ulcers are scattered over the inflamed and thickened mucous membrane. When the disease becomes chronic, the necrotic areas are completely replaced by ulcers, which may be so extensive that only scattered islets of thickened mucous membrane remain.

Symptoms.—The incubation period is between one and seven days. The disease generally begins acutely with griping abdominal pain; this is soon followed by the passage of fluid stools containing mucus, which may be blood-stained from the onset. In the early stages between six and thirty stools, many of which consist entirely of mucus mixed with pus and blood, are passed in the twenty-four hours. When the rectum is involved tenesmus occurs, in addition to the abdominal pain which generally precedes defæcation; it may be so persistent that the patient has a constant desire to open his bowels and is unwilling to leave the bed-pan. In severe cases there is often incontinence of fæces. The anus may become inflamed, excoriated, and painful. Drinking, eating, and every movement intensify the desire to defæcate. Micturition may also be frequent and painful. The intestines are very rarely thickened, and there is not much tenderness, but the iliac colon is often palpable as a firm, rather tender contracted cord.

The patient rapidly becomes exhausted and greatly depressed. Moderate fever is generally present, and the temperature may rise as high as 105°. The pulse is rapid and weak.

In slight cases the severe symptoms do not last more than two or three days; in severer cases improvement only begins after a week or fortnight, and convalescence is slow owing to the great weakness which results from the diarrhœa and toxæmia. The

disease sometimes becomes chronic ; diarrhœa persists with the passage of blood and mucus, or in less severe cases of abundant thick mucus with little or no blood, and emaciation, weakness and anæmia become progressively greater for months or even years, though intermissions tend to occur in the more prolonged cases. In most chronic cases the patient ultimately recovers, but death may result from exhaustion.

In some epidemics, such as that which attacked the British troops in the South African War, arthritis involving one or more of the large joints may occur. The knee is most often affected ; there is a large effusion, but not much pain or tenderness. Complete recovery without suppuration always takes place, but sometimes not until several months have elapsed. This complication has been comparatively rare in the present war. I have seen two cases in which an attack of dysentery brought on acute gout in men who were already subject to the disease. It is also necessary to distinguish the arthritis which may follow the injection of serum from the dysenteric arthritis.

An individual, who has previously been subject to hæmorrhoids, is very liable to be troubled with bleeding and prolapse after an attack of bacillary dysentery ; the possibility of this source of hæmorrhage when the stools have become solid should be borne in mind.

Diagnosis.—The only conclusive evidence of the presence of bacillary dysentery is the discovery in the fæces of the bacillus with its characteristic cultural properties and its agglutination with specific immune sera. A piece of mucus free from fæces should be picked out of the stool and sent at once for examination, as the organism can be much more easily isolated from such a specimen than from the actual fæces. A definite result cannot be obtained in less than three days, but a presumptive diagnosis can often be made in thirty-six hours.

The blood of patients suffering from dysentery never agglutinates the bacillus before the seventh and sometimes not until the twentieth day, after which the reaction lasts for three months or more. It thus develops too late to be of much practical value, for specific treatment should be instituted at once in order to be successful. With Shiga's bacillus agglutination should be obtained

in a dilution of 1 in 50 and with Flexner's with 1 in 200 before infection can be regarded as certain; although a positive result is then conclusive, a negative result is of comparatively little value.

Bacillary dysentery differs from amœbic dysentery in the more acute onset, higher fever and severer toxæmic symptoms; tenesmus is more frequent and thickening of the colon is much less common; tenderness is less marked and when present is generally greater on the left side than the right. Severe hæmorrhage is rare in bacillary dysentery, and in chronic cases abundant mucus with little or no blood is more often passed than in amœbic dysentery. Hepatitis and hepatic abscess never occur, and local and general peritonitis are very rare. There is no leucocytosis, a greater number of leucocytes than 15,000 per c.mm. pointing strongly to amœbic dysentery.

In the epidemic in the Mediterranean Forces in 1915 the clinical picture was much confused by the frequent association of amœbic with bacillary dysentery. The possibility of such an association should be considered, whenever a case of apparently amœbic or bacillary dysentery does not respond to treatment with emetine or anti-dysenteric serum respectively.

In rare cases the onset is so acute and the stools are so watery and profuse that the disease may be mistaken for cholera, until large quantities of mucus with or without blood are also passed and the diagnosis becomes clear.

Prognosis.—Some epidemics appear to be due to a much more virulent strain of the bacillus than others, the mortality being occasionally as high as 40 per cent. Shiga's bacillus generally gives rise to a more severe and protracted form of dysentery than Flexner's bacillus. With the treatment described below the mortality is now often no more than 2 per cent.

Prophylaxis.—Vaccination against dysentery has not been much employed owing to the very severe local reaction, which follows the injection of killed cultures of the organism. The reaction is, however, said to be less severe if the bacilli are sensitised with an immune serum or treated with eusol, and it is probable that in the future a considerable degree of protection will be obtained by the use of polyvalent vaccines, made if possible from

strains isolated in the locality in which the vaccine is to be used. The general sanitary measures, as described for the prophylaxis of amœbic dysentery, are the only means of preventing the spread of the disease among armies in war-time.

In extensive epidemics it is impossible to deal with the carrier problem, but in small epidemics it would probably be wise to keep convalescent carriers under supervision until three negative examinations of the stools have been made at intervals of a week following the last positive result. An examination of the stools for occult blood—traces which can be recognised by chemical means though they do not colour the fæces—would also be of use by showing whether any actual ulceration is still present. The necessity for some such test, which would help to prevent a man being sent to duty before he had really recovered, was well shown in a convalescent patient, who was passing normally formed stools; as a little blood was still occasionally present I examined him with the sigmoidoscope and found numerous small superficial and readily bleeding ulcers, the intervening mucous membrane being abnormally thick and red. Some mucus removed directly from an ulcer contained Shiga's bacillus.

Treatment.—The specific treatment of bacillary dysentery with anti-dysenteric serum instead of emetine, the saline treatment, and the local treatment with enemata require special description, but otherwise the treatment is the same as for amœbic dysentery.

As the intestinal lesions are due not to the local action of the bacilli but to the action of their toxins, which are absorbed from the colon and cause necrosis of the mucous membrane on re-excretion, and as the general symptoms, including such complications as arthritis, are also due to the absorption of toxins, the rational treatment is to get rid of the toxins from the bowel as rapidly and completely as possible, and to neutralise the toxins which are absorbed by means of specific antitoxins. The former indication is met by treatment with saline aperients, and the latter by the use of anti-dysenteric serum.

Saline Treatment.—Saline aperients have been used for dysentery since the seventeenth century; they were successfully employed in the American Civil War and in the South African War,

but were found of no value by many Anglo-Indian physicians. This difference of opinion is now known to be due to the fact that the treatment is only of value in bacillary dysentery, which is less common than amœbic dysentery in most parts of India.

Half an ounce of sodium sulphate in saturated solution should be given at the onset. This is followed by drachm doses every four hours during the first day and then every six hours until the dysenteric character of the stools disappears. If the patient is not seen on the first day the large initial dose should be omitted, and if the excessive action of the bowels has already resulted in exhaustion, no saline aperient should be given at all. This treatment not only exerts a favourable influence on the character of

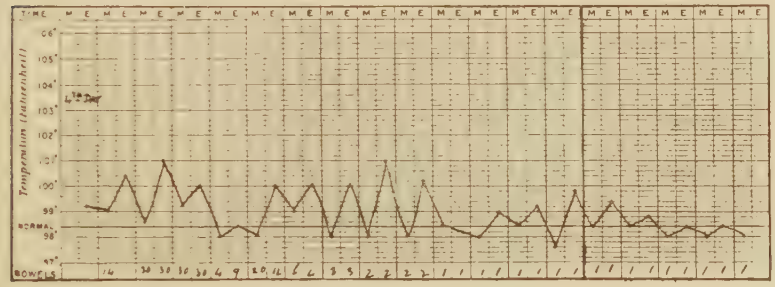


CHART I.

Bacillary dysentery treated with salts but no serum.

the stools, but it also relieves tenesmus. In mild cases the saline treatment often results in a rapid recovery (Chart I.), but there is considerable risk that the patient will remain a carrier, who is likely to have a relapse with any accidental irritation of his bowels and is at the same time a source of infection to other members of his unit. For this reason the serum should be given in mild as well as severe cases if a sufficient quantity is available.

Serum Treatment.—As the serum of horses immunised against one variety of dysentery bacillus is only useful in the treatment of dysentery due to infection with this variety, a polyvalent serum prepared by injecting horses with all available strains of the dysentery bacillus should be used ; a serum of this kind is made by the Lister Institute. In severe cases it should be given as soon as the patient is seen, if the dysentery is probably

bacillary in origin ; in mild cases the injection may be postponed until bacteriological confirmation is obtained.

The serum should be injected intravenously, as the antitoxin is then immediately available to neutralise the toxin, whereas if subcutaneous injection is employed, a delay of some days occurs before the whole of the serum is absorbed ; serum given by intramuscular injection is absorbed rather more rapidly than when given subcutaneously.

In asthmatics and in patients who have on any previous occasion received dysentery, diphtheria or tetanus antitoxin or other serum the danger of anaphylaxis can be overcome by a preliminary subcutaneous injection of $\frac{1}{10}$ c.c. of the serum half an hour before the first large injection, and by giving the latter

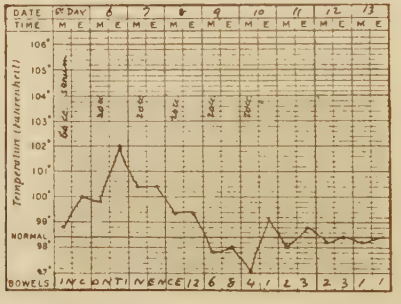


CHART II.

Bacillary dysentery treated with intravenous injections of serum.

into a muscle instead of into a vein. The danger of anaphylaxis is not merely theoretical ; in July, 1916, a man died on a hospital ship a few minutes after 20 c.c. of antidysenteric serum had been injected intravenously ; he had had three previous injections without ill effect, the last having been at Salonica 24 days earlier. He felt "funny" and became pale during the injection ; his whole body then became deeply flushed ; in a few minutes the flushing was replaced by extreme cyanosis, his pulse became weaker, and he died in about a quarter of an hour.

In mild cases 40 c.c. of serum are sufficient, but in very severe cases as much as 100 c.c. should be injected at one time : 60 c.c. given on the first day, followed by 20 c.c. on the two following days, is an appropriate dose for cases of moderate severity. If

necessary, however, the dose may be repeated daily for ten or more days. When hæmorrhage is severe the serum has the additional advantage of increasing the coagulability of the blood; no doubt it is this property which explains the good effects occasionally observed when the serum has been given in amœbic dysentery, but in such cases ordinary horse serum would be equally efficacious.

Technique of Intravenous Injection.—The serum should be warmed by standing the bottle in water at 100° F. If a deposit is present, the serum should be drawn off in such a way that the deposit is left with the last few drops in the bottle. The injection should be made with a sharp needle into one of the large veins of the elbow, after the skin has been cleaned and painted with iodine, and the veins have been made prominent by tying a bandage round the arm or by simply grasping the arm with the other hand. The serum is drawn into the syringe, which should not be completely filled, and all air bubbles should then be removed: the needle is inserted into the vein and a little blood is withdrawn into the syringe in order to be sure that the needle is within the vein: the bandage is then removed and the serum slowly injected. If a larger quantity has to be introduced than the syringe can hold, it is disconnected, the needle being left in position and the finger placed upon the vein whilst an assistant refills the syringe. In severe cases a funnel instead of a syringe should be attached to the needle, and the serum, together with two or three pints of saline solution, should be run in by gravity.

Local Treatment.—Astringent and antiseptic drugs given by the mouth are of little or no use, as they become too greatly diluted by the time they reach the colon, if given in doses sufficiently small to pass through the stomach and intestines without damaging them, and when they reach the affected part of the colon they are washed along at too great a speed to have any effect. The only method of treating the bowel locally is by enemata, which are extremely valuable in the chronic colitis and chronic diarrhœa, which frequently follow acute amœbic and bacillary dysentery, especially if specific treatment has not been given sufficently early and energetically. I have found tannic acid (gr. iv to the ounce) most useful of the numerous drugs which have been recommended, with the exception of silver-gelatose [albargin] (gr. i to the ounce),

which is, however, at present unobtainable. Injections should not be used in acute cases: quite apart from the difficulty in administering them caused by the great irritability of the rectum, which makes it impossible to retain fluid sufficiently long to be of any value, there is actual danger in severe cases; I know of one recent case in which the injection caused sudden violent abdominal pain owing to perforation of an ulcer, and death from general peritonitis followed three days later. One and a half pints of the fluid at 100° F. should be slowly run into the rectum from a funnel or douche-can, held not more than one and a half feet above the patient, the tube being introduced only just beyond the anal sphincter. The patient may lie on his left side, but the cæcum is reached most rapidly if he afterwards assumes the knee-elbow position for a few minutes. The injection should be given soon after the bowels have been opened, and the fluid should be retained for as nearly half an hour as possible, though it cannot as a rule be retained for more than a few minutes at first. The treatment should be repeated daily or on alternate days between three and five times; the series can, if necessary, be repeated after a week's interval.

(iii) Flagellate Diarrhœa and Dysentery

Lamblia intestinalis and *Trichomonas intestinalis*, flagellate organisms, were found in the stools of a small proportion of British soldiers, who contracted diarrhœa and dysentery in Egypt, Gallipoli, and Lemnos.

Lamblia inhabits the upper part of the small intestine, where it may become encysted and be passed in the fæces to gain access to the intestine of another individual, in which the active lamblia again develops. Numerous cysts are frequently found in the stools of healthy individuals living in the tropics, so that their discovery in a case of diarrhœa or dysentery does not prove that they are the cause of the symptoms; in almost all the cases of dysentery in the Mediterranean Expeditionary Force in which they were present, the *E. histolytica* or *B. dysenteriæ* was also found when the fæces were examined during the early stages. Possibly the changes in the intestinal contents caused by dysentery favour the multiplication of lamblia. Sometimes, however, attacks of

abdominal discomfort and diarrhœa with the passage of mucus containing enormous numbers of active lamblia occur; in such cases the organism is probably the cause of the symptoms.

Trichomonas is an inhabitant of the colon. It has never been seen in an encysted form and differs from other intestinal protozoa by surviving in fæces for a week or more in a spherical, but unencysted form, which becomes active again on warming. Its discovery has much the same significance as that of lamblia, as it is also frequently present in normal stools. It has been found in the drinking water during an epidemic of diarrhœa, and the purification of the water was followed by the disappearance of the epidemic. Only when it is present in excessive numbers in the stools of patients suffering from diarrhœa, is it likely to be the cause of the symptoms. The diarrhœa, which may persist in cases of dysentery after treatment with emetine or anti-dysenteric serum, is sometimes due to additional infection with lamblia, which is not cured by these means, though trichomonas is destroyed by emetine.

Treatment.—Turpentine (m. xv), methylene blue (gr. iii), and beta-naphthol (gr. xv), with bismuth salicylate (gr. xx), have all been recommended for the treatment of lamblia infection and should be tried in turn, as each succeeds in some cases, but fails in others. The treatment of trichomonas is identical with that of amœbic dysentery.

(iv) Ciliate Dysentery

Balantidium coli, a ciliate and much the largest intestinal protozoon, is a common inhabitant of the intestine of pigs. It escapes in an encysted form and may then gain access to the human colon. It has been found in the stools of normal individuals and in rare cases of dysentery in Russia, Norway, Sweden, and America. It was present in a few cases of dysentery occurring in British troops in Egypt in 1915. It produces lesions in the colon and liver identical with those produced by the *Entamœbia histolytica*.

Symptoms.—The symptoms are similar to those of amœbic dysentery, but fewer stools are passed, and anæmia is more marked owing to the hæmolytic action of a toxin produced by the

parasite in addition to the loss of blood in the stools. There is no leucocytosis.

Treatment.—Emetine is useless, but the administration of thymol causes numerous dead balantidia to be passed, and in mild cases the symptoms may disappear. Two cachets, each containing gr. x of thymol, are given every hour for four hours; two hours after the last dose a drachm of sodium sulphate is given. Alcohol and all fat-containing food must be avoided until the aperient has acted; otherwise thymol, which is dissolved by alcohol and fat, may be absorbed and give rise to serious toxic symptoms. Rectal injections of tannic acid (gr. iv to the ounce) should also be given, as this drug destroys any balantidia which are free in the lumen of the bowel.

Note on the treatment of Diarrhœa during Dysentery Epidemics

As an attack of diarrhœa may be due to irritation of the bowels by improper food or by sand, an aperient, preferably an ounce of castor oil with Tinct. Opii mx, should be given at once: this may also get rid of the earliest infection with specific organisms. The patient should keep as quiet as possible and have the lightest and most digestible diet available. If amœbic dysentery is prevalent, a grain of emetine hydrochloride should at once be injected, even if no blood or mucus is passed, as the diarrhœa may be the first sign of amœbic infection. If improvement occurs, but the diarrhœa does not cease completely, an additional dose of half a grain should be injected on the two following days. Some opium preparation may be given if the slight diarrhœa and colic continue, but the dose should be insufficient to cause constipation. If in spite of this treatment the diarrhœa persists for a fortnight sufficiently to cause general unfitness, or for a week if at all severe, the patient should be sent into hospital.

If at the onset of the diarrhœa or during the preliminary treatment any blood or mucus is passed, the temperature is raised to 100° or more, or tenesmus or severe colic occurs, the patient should be kept warm and at rest, and a grain of emetine should be injected when amœbic dysentery is the prevalent form, the dose being repeated daily for three days. If marked improvement does not

occur, or if the symptoms are severe from the onset, the patient should be sent to hospital at once. If the prevalent type of dysentery is bacillary, and amœbic dysentery is either absent entirely or rare, no time should be lost in trying emetine, but the patient should be sent to hospital for treatment with anti-dysenteric serum after receiving a preliminary dose of half an ounce of sodium sulphate.

For efficient early treatment it is thus essential that the type of dysentery prevalent in each part of the Army should be made known as widely as possible, and that the discovery of a change in type should be promptly notified to all regimental medical officers, as well as to hospitals which have no bacteriological laboratory of their own, as it is quite impossible for pathological examinations to be made of the fæces in more than a small proportion of cases during the first few days of the illness.

References

Sir Leonard Rogers: *Dysenteries*, London, 1913, and *British Medical Journal*, 1913, II. 1198.

L. P. Phillips: *Amœbiasis and the Dysenteries*, London, 1915.

A. Balfour: *Journal of the R.A.M.C.*, xxv. 473, 1915.

C. M. Wenyon: *Journal of the R.A.M.C.*, xxv. 600, 1915.

Sir Ronald Ross: *Proc. Royal Society of Medicine*, ix. 73, 1915, and *Lancet*, 1916, I. 1.

W. J. Penfold, H. M. Woodcock, and A. H. Drew: *Brit. Med. Journ.*, 1916, I. 407 and 714.

R. G. Archibald, G. Hadfield, W. Logan, and W. Campbell: *Journal of the R.A.M.C.*, xxvi. 695, 1916.

L. S. Dudgeon: *Proc. Roy. Soc. Med.*, "Occasional Lecture," March 8th, 1916.

CHAPTER III

AMŒBIC HEPATITIS AND HEPATIC ABSCESS

Etiology.—Amœbic hepatitis and amœbic abscesses of the liver are invariably secondary to amœbic dysentery. In 98 per cent. of fatal cases Rogers either obtained a history of dysentery or found amœbic ulcers or scars of ulcers in the large intestine. The patient had had dysentery or was still suffering from it in 72 per cent. of cases admitted into his wards with amœbic abscesses, and in an additional 14 per cent. there was a history of diarrhœa, which was doubtless a mild form of amœbic dysentery; in the remaining cases, in which no intestinal symptoms were recorded, amœbic ulcers had probably been present in the cæcum or ascending colon, situations in which they do not necessarily give rise to any symptoms Dysentery may precede the development of an abscess by months or even years, and when hepatitis occurs during an attack of dysentery, the latter generally becomes less severe as the inflammation in the liver progresses.

Chronic alcoholism is an important predisposing cause, but it is not essential, as the disease may occur in teetotallers. Exposure to sudden changes of temperature may bring on an attack, but it is important to avoid mistaking a rigor at the onset for the cause of the illness.

Considering the large number of cases of amœbic dysentery, which occurred in the Mediterranean Expeditionary Force, remarkably few soldiers have up to now developed amœbic hepatitis or hepatic abscess. This is partly due to the thorough treatment with emetine which the majority received. But the treatment was not always continued for a sufficient period, especially at the beginning of the epidemic, and a considerable number of slight cases must have escaped treatment altogether, so that there can be little doubt that hepatitis and hepatic abscess will frequently occur during the next few months and even during the next few years

among men who have served in Gallipoli or Egypt. A visit to the military hospitals in Malta with Colonel A. E. Garrod in June, 1916, convinced me that hepatitis, if systematically looked for, will be found to be a more frequent cause of chronic ill-health than has hitherto been recognised.

Pathology.—The amœbæ, which collect in the thrombosed veins at the base of dysenteric ulcers, pass to the liver by the portal vein and give rise to thrombosis in the portal radicles. The circulation is thus obstructed and necrosis of the surrounding tissue occurs; at the same time pus is secreted as a result of the irritant action of the amœbæ on the liver tissue. The abscess cavity gradually becomes limited by a fibrous capsule produced by the inflammatory reaction.

In 70 per cent. of cases a single abscess is present; in nearly half of the other cases there are two abscesses, in a quarter there are three, and in the remainder there are four or more. A single abscess is found five times as often in the right as in the left lobe of the liver.

The pus generally contains amœbæ, but no bacteria; in chronic cases all the amœbæ may have died, but they can still be found in the material obtained by scraping the wall of the abscess. In rare cases an amœbic abscess becomes spontaneously sterile, and its dried remains have been found at a post-mortem examination years afterwards. When an hepatic abscess reaches the surface, adhesions form between the liver and the adjoining structures, so that the contents of the abscess may burst into the lung, stomach or bowel, or open externally without infecting the serous cavities.

Symptoms.—The symptoms vary so greatly in different cases that it is convenient to describe separately the fulminating variety, in which the whole liver is riddled with small collections of pus with no fibrous tissue separating them from the surrounding liver substance, the common subacute form, and the very chronic form, but every gradation occurs between these three types. As most cases of the subacute type, including some with severe symptoms, subside completely with emetine injections, it is clear that in the early stages acute hepatitis without actual suppuration is present. The symptoms of subacute amœbic hepatitis and those of hepatic abscess can therefore be described together.

(i) *Fulminating Multiple Abscesses of the Liver.*—About one-tenth of the cases are of this type. There is always a definite history of dysentery, which is often still present when the hepatitis develops. The liver rapidly increases in size ; it is very painful and extremely tender. Slight jaundice may be present. The temperature is high with rapid remissions, the rise being often accompanied by rigors and the fall with copious sweating. Leucocytosis is well marked. Death generally occurs between six and eighteen days from the onset of symptoms.

(ii) *Subacute Hepatitis and Subacute Abscess of the Liver.*—Discomfort and a sense of weight are felt in the right hypochondrium in the slighter cases, but in the more severe ones the pain may be so great that the patient is hardly able to move, and he is often unable to lie on his left side owing to the dragging pain caused by the change in position. Pain may also be referred to the right shoulder and occasionally to the right arm, especially when the upper part of the liver is involved. When the left lobe is involved the pain may extend to the left side. The liver is enlarged and tender, but the rigidity of the abdominal muscles may be so great that it is impossible to feel its edge. Slight jaundice is occasionally present in the severer cases. The appetite is impaired or lost, and the patient rapidly becomes weak and emaciated.

The temperature is generally remittent, varying between 100° in the morning and 103° or 104° in the evening. In severe cases it remains high with only small remissions, but in more chronic cases, especially when the abscess bulges through the capsule of the liver and the tension within its cavity consequently falls, the fever is less marked ; it may then be low, continued, or relapsing, and may finally disappear. Copious sweating is common in the more severe cases. Moderate leucocytosis is always present, but there is less increase in the proportion of polymorphonuclear cells than in ordinary septic infections, the percentage being generally between 70 and 80.

The upper part of the liver is most frequently involved. The main increase in dulness is then in the upward direction, but the lower border is also abnormally low. The lower ribs bulge and the intercostal spaces become wider ; the skin may become œdematous, even in acute hepatitis without suppuration. At an early stage the breath sounds become feeble and the percussion

note impaired at the right base; the X-rays show that the right side of the diaphragm is abnormally high, and when suppuration occurs its movements are diminished and finally cease completely. If the diaphragm is perforated by an abscess of the liver, a pulmonary abscess or less frequently an empyema develops. In the former case large quantities of thick reddish pus are expectorated, and spontaneous cure often results; the sputum differs from that brought up in other forms of pulmonary abscess in being odourless. The base of the lung becomes dull to percussion and the vesicular murmur and voice sounds are much diminished and may disappear completely. The X-rays show a dense shadow in the lung, which cannot be clearly differentiated from the shadow of the liver.

An abscess in the anterior and lower part of the right lobe produces a tender tumour in the right side of the epigastrium; the lower ribs often become prominent and the lower border of the liver can either be felt or is found by percussion to be displaced downwards. A rub may be heard when an abscess reaches the surface of the liver, but it disappears on the formation of adhesions with the anterior abdominal wall. In advanced cases the skin becomes œdematous, fluctuation is present, and the abscess may open externally just below the costal margin. Less frequently the abscess reaches the under surface of the liver, when it may rupture into the duodenum or hepatic flexure of the colon; pus is then passed by rectum and the tumour suddenly diminishes in size; the general condition of the patient immediately improves, the temperature falls and spontaneous recovery may follow. In a case I saw at Salonica the rupture of a hitherto unsuspected abscess into the lung and intestine at the same time led to rapid recovery.

An abscess in the left lobe of the liver generally gives rise to a tender tumour in the epigastrium; if it becomes adherent to the anterior abdominal wall, definite fluctuation is obtained. An abscess bulging from the lower or upper surface of the left lobe is less easily diagnosed; it may escape recognition till it ruptures respectively into the stomach, when the characteristic thick reddish pus is vomited, or into the left lung or pericardium.

In some cases the X-rays show a localised increased density in the hepatic shadow, especially if the abscess is in the left lobe or if it is large and surrounded by a thick fibrous capsule. Localised subdiaphragmatic, subhepatic and retroperitoneal

abscesses are uncommon complications, and general peritonitis rarely develops.

(iii) *Chronic Hepatitis and Chronic Abscess of the Liver.*—After a period of alternating diarrhœa and constipation, sometimes with a definite history of dysentery, the patient feels ill, his appetite is poor, he loses weight and strength, and his temperature rises to about 100° every night. The area of hepatic dulness is increased, especially upwards, and the lower border may be felt below the costal margin. There is often a dull dragging sensation in the right hypochondrium, but there is little or no tenderness. The blood changes and the X-ray appearances are the same as with a subacute abscess. The nature of the disease may remain unrecognised until rupture into the lung, stomach, or bowel gives rise to characteristic symptoms.

Cirrhosis of the Liver.—According to Rogers the long-continued slight amœbic infection of the liver which may result from very chronic dysentery can eventually produce cirrhosis, which is for this reason very common in Calcutta and Egypt. Soldiers, who have had amœbic dysentery and have not been treated by emetine injections with sufficient thoroughness, will therefore perhaps be particularly liable to develop cirrhosis of the liver, if they indulge too freely in alcohol. In the future it will be wise to inquire whether men suffering from cirrhosis were in the Mediterranean Expeditionary Force, and if the answer is in the affirmative a course of emetine injections should be given.

Diagnosis.—The possibility of amœbic hepatitis or hepatic abscess should be considered whenever a soldier, who has recently served in India, Egypt, or Gallipoli, is suffering from progressive deterioration in health with more or less pyrexia, especially if the latter is remittent and accompanied by chills and sweats, and if obscure abdominal symptoms are present. A history of dysentery or simple diarrhœa, occurring in India or Egypt, or in Gallipoli during the summer and early autumn of 1915, would strengthen the suspicion, which would be further increased if no emetine or only one or two injections had been given, but the diagnosis would not be excluded in the absence of such a history. The most characteristic local symptoms are progressive enlargement of the liver, pain in the region of the right hypochondrium and in the right shoulder, and tenderness over the liver.

The condition is most commonly confused with malaria; but the rise of temperature is generally in the evening instead of during the day, and the liver is enlarged out of proportion to the spleen instead of *vice versâ*, leucocytosis is present with a relative increase of polymorphonuclear cells and only 2 to 4 per cent. large mononuclear cells in contrast with a normal or subnormal count with 15 to 20 per cent. large mononuclear cells; the malarial plasmodium cannot be found in the blood, and emetine gives good results whilst quinine fails to influence the pyrexia.

A localised tumour in the liver in a man who has been exposed to amœbic infection is generally an amœbic abscess; it is elastic or fluctuating, unlike the hard solid tumour formed by a gumma and a growth, which moreover are unaccompanied by marked pyrexia and leucocytosis. A suppurating hydatid cyst is a much less probable diagnosis in such an individual, and in most cases eosinophilia is present, though it is absent in amœbic hepatitis.

Cases of basal pneumonia or pleurisy, especially on the right side, occurring in a soldier who has recently served in India or the Eastern Mediterranean, should suggest the possibility of an amœbic origin, if anomalous symptoms are present, such as a gradual onset and prolonged course, absence of the characteristic rapid respiration, and irregular pyrexia with repeated chills.

Prognosis.—Until 1907 hepatic abscess was the second commonest cause of death in the British Army in India, but the incidence of the disease has become much smaller since amœbic dysentery has been treated by emetine injections, and the mortality has been greatly reduced since the necessity for operation has become comparatively rare owing to the frequency with which treatment is instituted in the pre-suppurative stage. The prognosis is always rendered worse if an open operation is necessary. It is best if the abscess is single, as multiple abscesses are difficult to locate and their evacuation may be impossible, especially in the rare fulminating cases. Abscesses which open into the lung, stomach, or bowel often get well spontaneously. The presence of active dysentery renders the prognosis worse, as further abscesses may develop and the patient is simultaneously weakened by the diarrhœa and loss of blood.

Treatment.—In acute hepatitis without suppuration very rapid improvement follows the subcutaneous injection of emetine hydrochloride in the same dosage as recommended for the treatment of amœbic dysentery: the hepatic tenderness diminishes within eight hours of the first injection, and the temperature may fall to normal in twenty-four hours. As there are no definite signs which distinguish hepatitis without suppuration from hepatic abscess, unless a definite tumour is present, no local treatment should be adopted until emetine injections have been given daily for a week without producing any improvement. There is some evidence to show that even a small abscess may be completely absorbed without aspiration, after the amœbæ in its walls have been killed by means of subcutaneous injections of emetine.

Hepatic abscesses were at one time often treated with a moderate degree of success by repeated aspiration. This treatment was gradually superseded by drainage after an open operation, but the results were not very good owing to the liability to secondary infection of the previously sterile abscess with pyogenic organisms. The most satisfactory treatment is that advocated by Rogers, in which the abscess cavity is evacuated by aspiration, and emetine is injected subcutaneously in order to kill the amœbæ in the abscess wall and in any ulcers which are still present in the colon. If the pus reaccumulates, a grain of emetine hydrochloride dissolved in an ounce of water is injected into the abscess cavity after it has been aspirated. Unless the situation of the abscess is already evident, repeated exploration under an anæsthetic is generally necessary before aspiration can be carried out.

Only in the comparatively rare cases in which bacteriological examination of the pus obtained by aspiration shows that the abscess is already infected with bacteria, is there any indication for an open operation and drainage.

When an abscess has ruptured into the lung, stomach, or bowel, subcutaneous emetine injections should be continued until the temperature has remained normal for a week. Recovery generally takes place without aspiration or drainage becoming necessary.

Reference

Sir Leonard Rogers: *Dysenteries*, p. 136. London, 1913.

CHAPTER IV

TRENCH FEVERS

After all examples of the well-defined acute infections had been separated from the cases of fever occurring in the British armies in France and in Salonica, a considerable number remained in which the diagnosis was doubtful. A few were probably due to infection with the *B. paratyphosus* or even the *B. typhosus*, although all bacteriological examinations had proved negative, but I do not think that these should amount to more than 5 per cent. of the cases in which bacteriological confirmation is obtained. The large majority are most frequently diagnosed as "pyrexia of unknown origin," influenza, and rheumatic fever, the first of these being the only diagnosis which is indisputably correct. A small number of cases of true influenza with the characteristic catarrhal and general symptoms undoubtedly occur, although an attempt is rarely made to confirm the diagnosis bacteriologically. Rheumatic fever is rare; Herringham only saw five cases in France between October, 1914, and October, 1915, though it subsequently became rather more common, and I only saw a single definite case in Salonica; in the few cases so diagnosed, in which the pain is actually in the joints and not in the muscles or tendons, the arthritis is more often gonococcal than rheumatic. We are thus left with a number of cases of pyrexia of unknown origin. In this chapter two varieties of a well-defined febrile disease are described, which account for a considerable proportion of these cases.

In the early summer of 1915 Major J. H. P. Graham drew attention to a type of fever occurring in the British Army in France, in which two periods of pyrexia were separated by a normal interval. Similar cases were recognised with increasing frequency, and the disease soon became widely known as "trench fever." In November, 1915, Captain G. H. Hunt and Major A. C. Rankin described thirty cases of the same type of trench fever,

and a still fuller account was published in February, 1916, by Captain J. W. McNee, Lieutenant A. Renshaw, and Captain E. H. Brunt, in which for the first time two distinct clinical types were distinguished.

The disease was only observed among officers and men living near the trenches, and in the personnel of hospitals, especially among orderlies of wards in which there were patients suffering from the disease. No cases occurred among ammunition columns, ordnance, and headquarters troops. It was for this reason that the name "trench fever" was adopted, though actual residence in the trenches themselves was certainly not an essential factor, and Hunt and McNee, in their most recent communication, state that cases have lately been met with further from the front.

According to Colonel Sir Wilmot Herringham, literally thousands of cases of the first type occurred among the troops in France and Flanders between the end of April and October, 1915; it was comparatively rare in the following winter, but increased again in the spring of 1916. It did not occur in Gallipoli and was not definitely recognised in Salonica until April, 1916. The second type, which was rare in France and Flanders until November, 1915, when it became more common than the first, has been very prevalent in Salonica since December, 1915. At first cases had to be recorded as "P.U.O." (pyrexia of unknown origin), or under the head of influenza or some other equally incorrect name, even when their nature had been recognised. For this reason it became customary to call the disease "pyrexia of unknown origin (A)," or shortly, P.U.O. (A), as this designation did not introduce into the official records an unauthorised name, such as "periodic one day fever" or "Salonica fever," which had been used by some medical officers. The term trench fever was not used, as besides being an unauthorised name, the disease was common in Salonica at a time when no men were living in the trenches, and cases occurred in ammunition columns several miles from the trenches as well as in front-line troops and in hospital orderlies. When, however, in March, 1916, we recognised that the disease was identical with the long form of trench fever described by McNee, Renshaw, and Brunt, and when a month later the common short form of trench fever seen in France and Flanders became prevalent in Salonica, it seemed wise to adopt this name.

A few cases with symptoms intermediate between the two types of trench fever have been observed in France and in Salonica, and the initial symptoms are very similar, but the temperature chart is so different in typical cases, and the second class occurred in such large numbers without the first in the winter months, although it was very rare in comparison with the first during the summer of 1915 in France, that it cannot yet be regarded as finally proved that the two diseases are really due to the same infection, as is generally believed by British observers in France.

My attention was first drawn to the disease in Salonica at the beginning of January, 1916, by Lieut.-Colonel D. J. McGavin, Major D. S. Wylie, and Major H. T. D. Acland, of No. 1 New Zealand Stationary Hospital. At first it was not widely recognised, but as medical officers became more familiar with its characteristics, it became clear that it was extremely common, especially in certain units.

With the exception of one man, who contracted the disease whilst in hospital from another cause, all of the cases observed in Salonica up to March, 1916, belonged to two divisions which had been several months in France. None had been in Gallipoli or Serbia. The short form of trench fever had occurred in several of the affected units whilst they were in France. It is highly probable that these divisions brought the infection with them. In March, 1916, the periodic form of trench fever began to occur in a division which had been in Serbia, but not in France; some of the men had been in Gallipoli, but most had come straight to Salonica from England. In April groups of cases of both forms of trench fever appeared in units belonging to other divisions, which had been free from the disease since their arrival at the end of 1915. The infection in the later cases was probably conveyed by men coming in drafts from France. The disease also developed among the personnel of some of the hospitals, to which the patients were sent. It has not been recognised among the French troops in Salonica, and does not appear to have been prevalent among them in France until July, 1916.

Trench fever has occurred among Austrian troops in the Tyrol and German troops on the eastern front; the short form was described in the *Wiener klinischer Wochenschrift* as gaiter fever, and the periodic form in the *Münchener medizinischer Wochenschrift* as five-day fever.

Pathology.—Before trench fever could be accepted as a clinical entity it was necessary to prove that it was not an aberrent form of some other condition, such as paratyphoid fever, which it may closely simulate during the first pyrexial period, true relapsing fever, which it resembles in so far as the fever is of a characteristic relapsing type, and malaria, which is sometimes simulated by the shorter and sharper pyrexial attacks. But the blood taken at the height of both the initial and the later febrile periods has always been sterile on cultivation, the Widal reaction has been consistently negative at every stage of the illness, and no spirochæte nor malarial plasmodium has been found, in spite of repeated examinations of blood films taken during both febrile and afebrile periods.

McNee and Renshaw found that trench fever could be transmitted to healthy soldiers by the intramuscular and intravenous injection of the blood of men suffering from the disease. Injection of the washed red corpuscles had the same effect, but the plasma and serum were not infective.

One attack does not seem to protect against re-infection. I saw a man with the long type of trench fever in April, 1916, in Salonica ; he had been seen by Captain McNee in a typical attack in September, 1915, while in France, and had been quite well in the interval. It is possible, however, that there was no re-infection, the original infection having remained latent between the two attacks.

As all attempts to discover the infective organisms have failed and as no fatal case has occurred, the nature of the disease remains unknown. The striking periodic character of the fever in the long type of case, the considerable increase in the proportion of large mononuclear leucocytes, which has been found on several occasions, and the evidence pointing to an intracorpuscular infection suggest a protozoal rather than a bacterial origin. Since this was written a spirochæte has been isolated from the blood.

Method of Propagation.—There is no nasal, pharyngeal or bronchial catarrh, and, except for constipation, gastro-intestinal symptoms, though occasionally well marked, are generally completely absent. It is probable, therefore, that the disease is not

conveyed by the respiratory secreta or by the faeces, but through the intermediation of some insect. The occurrence of the long form of the disease during the winter months shows that the infection can be conveyed in the absence of mosquitoes and other flying insects; though Herringham found mosquitoes in France throughout the winter, there were certainly none in Salonica. Fleas have been scarce in both countries, and the men themselves rarely complain of them. Almost all patients admitted that they were lice infested up to the time of their entry into hospital, so that it is quite possible that the disease is conveyed by lice. A hospital orderly, who had been free from lice since his arrival in Salonica, had to carry the kit of a number of new patients suffering from trench fever on May 2nd, 1916. The clothes were swarming with lice, and the same evening he found some in his own clothes. He got rid of them in the course of a few days, and on May 20th an attack of trench fever began. He was not employed in the wards, he never came in contact with any patients suffering from the disease, and he was the first case of trench fever in the personnel of the hospital to which he was attached.

The incidence of trench fever is least in the cleanest battalions and in the divisions which have the best facilities for bathing. In some units a successful campaign against lice has been immediately followed by a great diminution in the incidence of trench fever, and the campaign against lice in the whole Salonica Army in the spring and early summer of 1916 was followed by the almost complete disappearance of the disease. Captain A. L. Urquhart developed the short form of trench fever after allowing the lice from a patient with this form of the disease to bite him, and Captain McNee tells me that his observations in France have led him to agree with the conclusion I came to in Salonica—that the disease is spread by lice.

Cold, wet, and fatigue appear to be exciting causes in a man who has become infected, but has so far had no symptoms; thus Captain Hay noticed that almost all cases in his regiment in Salonica began two or three days after they had been wet through. In several instances a group of men sleeping in the same tent have been affected.

Some patients appear to be carriers, who do not lose the

infection completely for several months, but have recurrences from time to time, during each of which they may infect an additional number of men. A sergeant, who had been in good health whilst in France between December, 1914, and November, 1915, developed the periodic form of trench fever early in December, 1915, directly after he left France for Salonica ; in the following four months he was in hospital five times for a week or more, though he was perfectly well in the intervals. Every time he returned to his unit he became lice infested again, and he appeared to infect most of the men with whom he came in contact, about forty men of his company, including six sergeants, having been taken ill with trench fever between January and March ; one of the sergeants had wrestled with him, another had danced with him, a corporal slept next to him, and a private sat next to him for some lectures.

Incubation Period.—As a result of observations in Salonica on cases arising in hospital, when a patient had been admitted for some other condition, I came to the conclusion that the incubation period is between 15 and 25 days ; in the case of the hospital orderly already described it was probably 18 days. Quite independently Hunt and McNee in France concluded that it was between 14 and 24 days. The following three cases are typical of those which led to my estimate of the incubation period :—

(1) Sergeant B., R.E., was admitted for rhinitis on December 27th, 1915, into a ward in which there were at the time two patients suffering from the periodic form of trench fever, no other cases of which have yet been observed in his unit. On January 1st, 1916, he was moved into another ward, in which there were and have since been no such cases. When convalescent from the rhinitis, which had been accompanied by no pyrexia or pains in the head, back, or legs, he suddenly became ill on the evening of January 24th ; his temperature in the morning was normal, but at 6 p.m. it was 104°. This proved to be the first pyrexial period of a typical attack of the periodic form of trench fever. It is probable that the infection was contracted from the other cases in the ward between December 27th and January 1st, between 23 and 27 days before the onset of symptoms.

(2) Private W. went to France at the beginning of the war

with the 1st —— Regiment. He was wounded in January, 1915, and was in England until the end of 1915, when he came to Salonica, joining the 2nd —— Regiment, which had come there from France in November, or January 13th, 1916. A few days after he arrived he became lice infested. On January 21st he went to a field ambulance and then to a casualty clearing station with a hydrocele; he was transferred to a stationary hospital on February 6th. On February 12th his temperature rose and a typical attack of the periodic form of trench fever began. His clothes were disinfected when he entered the casualty clearing station, and he had no more trouble with lice after his admission there. It is probable that he contracted the disease whilst with his regiment, *i.e.* between 24 and 16 days before the onset; as he was not lice infested until he had been with his regiment some days, the period was probably about three weeks.

(3) A third patient was admitted into hospital for quinsy. He was in a ward, in which there were no other cases of trench fever, but he developed the disease 14 days after admission. He had probably contracted it whilst still with his regiment, in which at least one case had already occurred, so that the incubation period was over a fortnight.

Symptoms.—The disease generally begins suddenly without any premonitory symptoms, but a feeling of malaise occasionally precedes the attack for a day or two. The patient complains of severe headache, especially frontal and behind the eyes, and this is rapidly followed by pain in the lower part of the back and on the second or third day in the legs. Pain in the neck is occasionally observed; in two cases mentioned by Hunt and McNee pain and stiffness in this region were so severe that a lumbar puncture was performed in order to exclude meningitis, and I also saw two cases in Salonica in which this was done. The patient generally shivers, but there is never a definite rigor; he is occasionally flushed and often sweats profusely. The bowels are regular or constipated, and there is no nasal or bronchial catarrh; the appetite is lost, the tongue is moist and often slightly furred, and occasionally mild pharyngitis is present. Herpes labialis has occurred in a few cases, but less frequently in the long than in the short form of the disease. There is no rash.

The onset is sometimes extremely abrupt; the patient suddenly feels giddy, his legs give way under him and he shivers; he may be very short of breath, and occasionally complains of a pain in his left side. He has to fall out if on parade or marching, and has often great difficulty in returning to camp without assistance.

In a few cases, in which constipation is generally present, there is some abdominal pain with slight distension and tenderness, and there may be nausea and vomiting at the onset. Four out of my first fifty cases of the periodic type were sent to hospital diagnosed as appendicitis; in one a normal appendix was removed, and a second would have been operated upon had he not refused. The abdominal symptoms rapidly disappeared, and in the relapses they were less prominent than the other symptoms.

When the pain in the legs is severe, there may be some cutaneous hyperæsthesia over the shins. The shins are always tender, even if the patient complains of no pain in the legs, but tenderness appears to be most marked in groups of cases and at certain times. Graham did not mention it, and it was not observed in the earliest cases in Salonica. In several cases the periosteum of the tibia has seemed to me to be rough and thickened, as it pits slightly on pressure, although no pitting of the subcutaneous tissue was present. The tenderness is most marked over the lower half of the shins and may be very severe, a comparatively slight pressure causing the patient to cry out, and the pain produced may last for hours. A less degree of tenderness is often present in the tendons behind the knee, and occasionally in the ligamentum patellæ and along the course of the femur. In one case observed by Captain J. A. Gunn there was marked tenderness of the ulna on both sides. There is little or no tenderness of the calves or other muscles. The knee and ankle jerks are normal, and there is no evidence of neuritis, though a considerable degree of muscular atrophy may occur.

In the first attack the spleen is sometimes palpable or is found to be enlarged on percussion, and there may be some tenderness in the left hypochondrium. Although this was certainly the case in Salonica, Herringham, Hunt and McNee never found any splenic enlargement in the cases they observed in France.

Leucocytosis is often, but not always present during the pyrexial attacks; the count varied between 4,700 and 22,000 per c.mm. in 35 cases, mostly of the short type, examined in France (Hunt and Rankin; McNee, Renshaw, and Brunt); in many of the cases examined both in France and in Salonica there was a relative increase in the large mononuclear cells (Elworthy, Urquhart). Polychromatophil cells above the normal size with well-marked punctate basophilia were observed by McNee, Renshaw, and Brunt in France; but Elworthy, working in Salonica, came to the conclusion that they only occurred in the later stages of the more severe cases. The percentage of hæmoglobin is

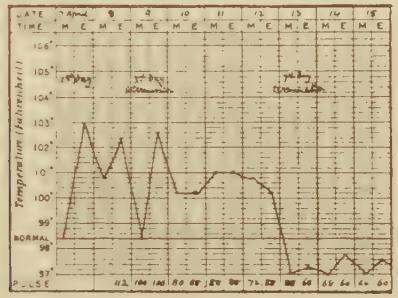


CHART III.

Short form of trench fever.

generally about 80, though the number of red corpuscles is undiminished.

In the *short form of trench fever* the temperature rises rapidly to between 102° and 104°, but the pulse rate is only slightly increased. On the third or fourth day the temperature suddenly falls—generally to normal or subnormal, but there is no corresponding improvement in the symptoms. After an interval of a few hours it rises again, and then after another two to five days it falls to normal; on this occasion there is immediate relief to all of the symptoms (Chart III.). In some cases the remission on the third or fourth day does not occur, the temperature remaining raised for about a week. There is often a single relapse after an interval varying from a few hours to ten days, but generally less than four days; the temperature rises to 100° or 101° for 24

or 48 hours, during which the symptoms return with diminished severity (Chart IV.). The patient is generally fit for duty almost immediately after the temperature falls again. Many cases have been kept under observation by Hunt and McNee for weeks or months after the fall of temperature without any return of fever or other symptoms, so that there could be no question of additional relapses occurring after the patient had been discharged from hospital.

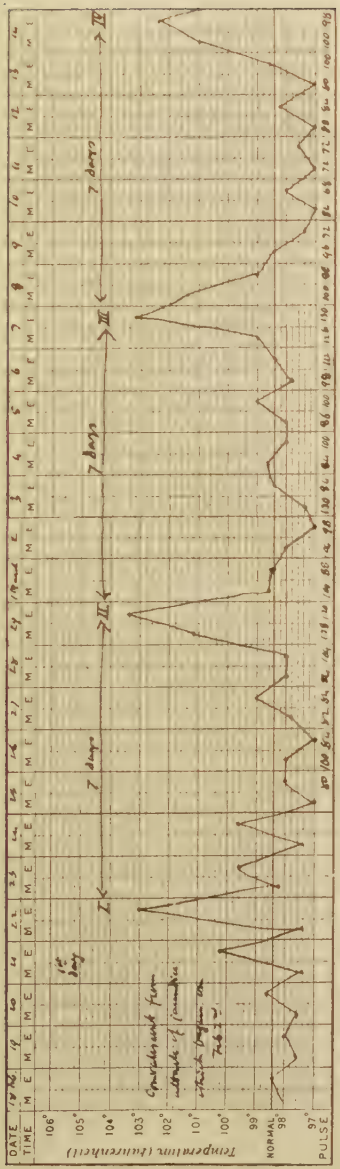
In the *long* or *periodic type of trench fever* the temperature rises to between 101° and 104° on the first evening. The initial attack is variable in duration; the temperature may be normal the first morning, high in the evening, normal the second morning,

CHART IV.

Short form of trench fever.

and rather less high the second evening than the first, after which it remains down. In other cases the first attack may last as long as four or five days, the temperature being always lower in the morning than the preceding and following evening, the highest temperature being reached on the second or third day; in one case it reached 105·8° on the third evening, though it was normal the previous and following mornings. The pulse is generally accelerated in proportion with the temperature, but at first it may be considerably faster. With the fall of temperature at the end of the initial attack all the symptoms disappear and the patient is often sent back to duty.

After being well for two to ten days he complains of a return of headache and pain in the legs, which culminate at night; the



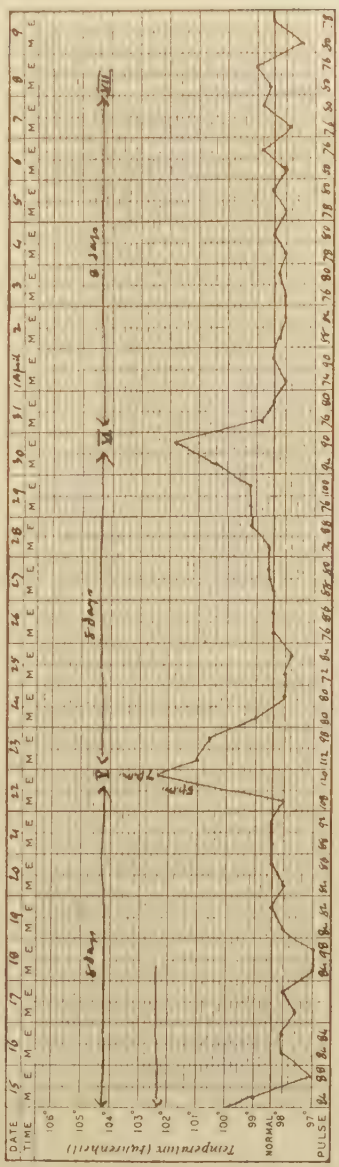


CHART V.

Periodic form of trench fever, developing whilst the patient was in hospital.

temperature rises in the evening to a point, which is generally a little lower than the highest temperature in the first attack. The temperature falls to normal or nearly normal the next morning, and either remains down or rises to a less extent the second evening, thereafter to remain normal. The general symptoms are much less severe than in the first attack, and the acceleration of the pulse is less marked, but the pain in the legs and tenderness of the shins are generally greater, and they may not disappear completely in the interval between the second and third attacks, though the headache, which generally remains the most prominent symptom during the attack, is never present in the apyrexial periods. The pain in the legs is sometimes extreme and may prevent sleep; in other cases it is comparatively slight and the patient looks and feels remarkably well, considering that he has a temperature of 101° or more.

Recurrences follow periodically, the maximum temperature being always reached in the evening (Chart V.). The intervals between the attacks are fairly constant in each case, but it varies in different cases between four and eight days, five being the most common interval. Each succeeding attack is generally milder than its predecessor and the temperature is rather lower, but in severe cases the patient feels weaker in the later intervals, and the pain and tenderness of the legs are more persistent. The later attacks may be of such short duration that the rise in temperature is not recorded at all if it is only taken twice a day (Chart VI.). On the afternoon and the evening of the day on which the attack is expected the temperature should therefore be taken every two hours, especially if there is any pain in the head or legs, as in most cases the patient knows from his sensations that there is going to be a relapse, even before the temperature rises. The temperature is sometimes only raised for three or four hours: in one case, for example, the morning temperature was 98°; at 5.30 p.m. it was 99°, at 6.30 p.m. 100°, and at 8 p.m. 101°; at 9 and 10 p.m. and at 8 a.m. the following morning it was 98·4°. In another case it was 97·6° at 5 p.m., though the patient had had a headache since the morning, but 101·2° at 8 p.m., 102·4° at 10 p.m., 101·4° at 2 a.m., 100·2° at 6 a.m., and 98° at 8 a.m., so the morning and evening chart showed no rise, as the temperature in the

ward was taken at 8 a.m. and 5 p.m. This liability for the rise in temperature to escape recognition accounts for the fact that it may appear from the chart that an attack has been missed, the interval between two of the later attacks being double that between the earlier ones: a headache may have been felt and a rise in the pulse rate recorded halfway between the attacks. In one case the third relapse was of exceptional duration and severity; this may have been due to a relapse having been really missed, as the apyrexial period which preceded it was of double length.

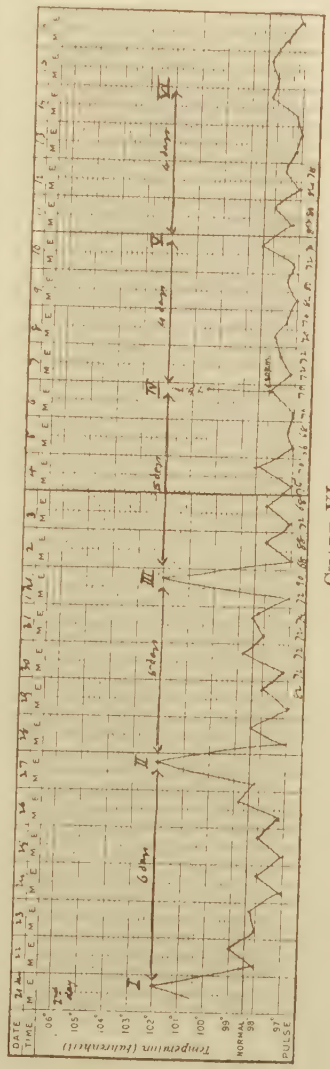


Chart VI.

Periodic form of trench fever; the fourth rise of temperature only occurred after the evening temperature had been recorded.

In a few cases the temperature remains raised for three or even four days in each attack, the evening temperature being always higher than the morning temperature, which may be normal on the first and last days; the highest point is generally reached the second evening.

Diagnosis.—The diagnosis can only be made with certainty from a study of the temperature chart, but the association of pyrexia with tender shins is very suggestive of trench fever already in the first attack. Painful and tender shins have, however, sometimes been observed in the Salonica Army in the apparent absence of fever, and the unsatisfactory name of "trench

shin" has sometimes been used to describe such cases. It is, however, not improbable that slight initial pyrexia whilst the patient was still at duty escaped notice. Some of the cases regarded as examples of the short form of trench fever are probably really periodic cases, as there is no doubt that the later bouts of pyrexia are often entirely missed owing to the short time they last, the patient having meanwhile gone back to duty, or if in hospital the evening temperature may have been taken at 5 p.m., although the rise only began at 7 p.m. or later. Several medical officers, who were very familiar with the early stages of the disease, only recognised the periodic rise of temperature after their attention had been specially drawn to its occurrence, as their patients had returned to duty after the first or second attack and had not "gone sick" for the later and comparatively slight recurrences.

The majority of cases were at first diagnosed as *influenza*, though it was generally recognised by medical officers that they were not identical with the familiar forms of that disease. Thus there is never any nasal or bronchial catarrh, the patient rarely appears or feels seriously ill, except sometimes during the first two days of the first attack, and respiratory and nervous complications never occur. The periodic return of pain and pyrexia and the pain and tenderness of the shins are quite characteristic, and prevent confusion with influenza except at the onset.

The possibility of *malaria* must always be considered, and a blood film should be examined for the malarial plasmodium before making a definite diagnosis in cases of doubt, especially if the patient has previously had malaria, or when it is prevalent, as was the case during the summer in Salonica. The differential leucocyte count is of no assistance, as there is a relative increase in large mononuclear cells in both diseases. The longer intervals between the attacks, their invariable occurrence in the evening instead of at various times of the day, the absence of true rigors and the failure of quinine given by mouth to modify the course of the illness are distinguishing features of trench fever. Several old soldiers at first thought that they were suffering from malaria, but they subsequently realised that the disease must be different, as they never before had had severe pain and tenderness in their shins.

A few cases of true *relapsing fever* occurred in British as well as Indian troops at Gallipoli, but only eight Indian and no British soldiers had been attacked by the disease in Salonica up to the end of April. The disease was, however, common in the Serbian Army in 1915, and was actually first described by Hippocrates in the Greek island of Thasos, so it is necessary to be on the look out for it among British as well as Indian soldiers at Salonica, and at least one case occurred in June, 1916. In six cases I saw with Captain F. F. Strother Smith, I.M.S., the pyrexial period was generally longer, varying between $2\frac{1}{2}$ and 6 days, and the fever was higher than in trench fever, the maximum temperature in each attack being between 104° and 105·6°; successive relapses did not diminish in severity unless they were cut short by injecting salvarsan, the effect of which was almost instantaneous; the patients were extremely ill and often delirious during the pyrexial periods, and the delirium occasionally continued after the temperature fell; bronchitis was common, and there was no pain in the legs nor tenderness of the shins; the apyrexial periods varied between two and eight days, six days being the most common, but the periodicity was less regular than in the periodic type of trench fever. The spirochæte was found without difficulty in the blood during the pyrexial period in every case. The disease may, however, be less severe when it occurs among Europeans.

Prognosis.—There have been no fatal cases and the patient never appears seriously ill, except occasionally for a very short time in the first attack.

Until the commencement of the hot weather in Salonica at the end of May no complications had been observed with the exception of phlebitis of the femoral vein in one case, and slight jaundice in two cases, but the latter at any rate was probably accidental. With the onset of the hot weather it was found that trench fever was often accompanied by a moderate degree of cardiac dilatation, which resulted in the development of "soldier's heart" if the patient returned to full duty too soon. Endocarditis has never occurred. Hunt and McNee have not observed albuminuria, but Herringham found a trace of albumin, which soon disappeared, in a few cases. I saw one similar case

in Salonica and another—a man invalided from France—with Captain W. R. Reynell.

The total duration of the periodic type of trench fever from the onset to the end of the last attack is generally between four and six weeks, but some cases appear to abort, and in a few others attacks may recur for several months, the patient remaining quite well in the intervals. I saw a sergeant with the periodic form of trench fever in January, 1916, in Salonica; he had had similar attacks at intervals since August, 1915, when he first became ill in France. In most cases the patient rapidly gets strong again after an attack and is generally fit for duty after the second period of pyrexia, though he may have to rest for a few hours when the later attacks occur. Sometimes, however, great exhaustion follows and convalescence is slow.

Prophylaxis.—As the disease is probably conveyed by lice, which become infected by biting a patient during an attack, every effort should be made to keep troops free from them. All cases of trench fever should either be sent to hospital or isolated, and the patient's clothes and bedding should be specially disinfected, as well as that of all men who have recently slept near him. After the initial or the second attack a man is often able to return to duty; it is very important that he should be kept under observation, and if he again becomes verminous his clothes and bedding should again be disinfected; men who are still having attacks or have recently recovered should sleep together, isolated from the other men in their unit, but there is no reason why they should not work with them.

Treatment.—No treatment has yet been found which prevents the periodic return of attacks or which is really effective in overcoming the pain. It is generally agreed that in the first attack considerable relief occurs if the tendency to constipation is prevented by aperients. Acetyl-salicylic acid is the most effective analgesic drug. Quinine given by mouth has very little effect, but in some cases I have found that the subcutaneous injection of ten grains of the bihydrochloride at the height of an attack, especially if repeated in three hours, appeared to prevent the occurrence of relapses. Salvarsan and antimony have proved useless.

Numerous local applications, both hot and cold, have been used for the painful shins: some of them have appeared to do good in certain cases, but the most frequently successful seems to have been a cold compress of saturated magnesium sulphate solution, which was first recommended by Captain D. S. Harvey. In slight cases gentle massage has given temporary relief, and I have seen a number of cases in which Captain W. R. Reynell has given galvanic baths for the legs with frequent reversals of the current with unexpectedly good results. In a few cases in Salonica the periosteum was incised, but when this was done on one side only, improvement occurred with equal rapidity on the opposite side. Even if the results had been more promising, I should regard the operation as quite unjustifiable, as the pain always disappears spontaneously in the course of a few weeks and often quite rapidly, even when it is exceptionally severe.

References

J. H. P. Graham: *Lancet*, 1915, II. 703.

G. H. Hunt and A. C. Rankin: *Lancet*, 1915, II. 1133.

J. W. McNee, A. Renshaw, and E. H. Brunt: *British Medical Journal*, 1916, I. 225.

W. P. Herringham: *Quarterly Journal of Medicine*, IX. 429, 1916.

G. H. Hunt and J. W. McNee: *Quarterly Journal of Medicine*, IX. 442, 1916.

H. Schrotter: *Wiener klinischer Wochenschrift*, XXIX. 197, 1916.

H. Werner: *Münchener medizinischer Wochenschrift*, LXIII. 402, 1916.

CHAPTER V

PARATYPHOID FEVER

Paratyphoid fever is a septicæmia caused by infection with the *Bacillus paratyphosus A* and *Bacillus paratyphosus B*, and in rare instances by other allied organisms. In morphological and cultural properties these bacilli closely resemble the *B. typhosus*, but they differ so decidedly from the latter and from each other in their reactions with certain media and in their behaviour with specific immune sera that they must be regarded as distinct and specific organisms.

Paratyphoid fever was first recognised clinically by Achard and Bensaude of Paris in 1896. Though paratyphoid B is probably more common in England than has hitherto been supposed, it is very rare compared with typhoid fever among the uninoculated general public, and it has never been observed in India ; its chief source in France appears to have been the civil population close to the front and especially the Belgian refugees. Some French observers, however, believe that the reoccupation after the battle of the Marne of territory, which had been fouled by paratyphoid carriers among the German invaders, was the primary source of the epidemic. Paratyphoid A is well known in India, though it had never been recognised in Europe until the present war ; the majority of the earlier cases which occurred in France were among troops from India or men who had been attached to such troops. From its subsequent widespread distribution, however, it seems probable that the disease must have been endemic, though hitherto unrecognised, in the Mediterranean area and perhaps also in France and Flanders.

Up to November, 1916, 1684 cases of typhoid fever and 2534 of paratyphoid fever had occurred among the British troops in France and Flanders, the number of cases of paratyphoid B

forming at first three-quarters and later only two-thirds of the total of A and B. The incidence of the disease steadily diminished after the first four months in spite of the increasing size of the army, and in November, 1915, there were only thirty cases of typhoid and ninety-two of paratyphoid fever in hospital in France—the lowest number since October, 1914. The disease was considerably more prevalent in Gallipoli and Lemnos, where it was probably ten times as common as typhoid fever; according to statistics published by Martin and Upjohn, about 5700 cases of enteric fever occurred among the 96,683 medical casualties, which were stated by the Under-Secretary for War to have occurred in this area up to the middle of December, 1915, and of these about 93 per cent. were paratyphoid and only 7 per cent. typhoid fever. Paratyphoid B was the prevalent type up to the end of October, when paratyphoid A became more common, and by December it had almost entirely replaced paratyphoid B; paratyphoid A was also very much more common than paratyphoid B in Salonica, though the total incidence was small.

Etiology.—Infection with paratyphoid fever occurs in the same way as with typhoid fever, the primary source being a patient suffering from the disease or a carrier who has had the disease, as the *B. paratyphosus* may still be present in the fæces a year after the illness, water or food becoming contaminated by his excreta directly, or more often indirectly by flies and dust. The sputum has been found to contain the organism in cases complicated by broncho-pneumonia; it must therefore be regarded as a possible source of infection to nurses and orderlies and to other patients in the ward.

Morbid Anatomy.—There does not appear to be anything about the morbid anatomy of the paratyphoid fevers, which distinguishes them from typhoid fever or from each other. There is perhaps a tendency both in paratyphoid fever A and B for the colon to be more widely involved compared with the small intestine than in typhoid fever, as ulcers are often present from the cæcum to the splenic flexure, and I have even seen them in the rectum, but this is not a constant feature, for in two cases the small intestine was alone infected and in a third—a case of paratyphoid A—no ulcers were present in any part of the inflamed intestines.

Carles refers to six similar cases recorded by French observers, and to one in which the intestines were not even inflamed.

Symptoms.—The symptoms of paratyphoid fever A and B are so much alike that they are best described together. The incubation period is between ten and fourteen days. The onset is more or less acute. The patient always complains of headache, often of abdominal discomfort, and sometimes of diarrhœa; the diarrhœa soon ceases, but the headache persists and pains in the back and limbs develop, so that by the fourth day he generally feels too weak and ill to continue with his duties. In some cases severe headache, abdominal pain with or without diarrhœa, and repeated shivering attacks develop so acutely that in a few hours the patient is obviously ill. Epistaxis, so slight that the patient generally does not mention it unless specially asked, was a common early symptom in France, but was comparatively rare at Lemnos and Salonica.

The headache may be sufficiently severe to prevent sleep the first few nights, after which it gradually improves. Diffuse abdominal discomfort is common; it rarely amounts to pain and generally only lasts two or three days, but in very acute cases it may be severe and accompanied by vomiting. The tongue is furred, and in severe cases it is dry and brown; if an entirely fluid diet is given, it remains coated except at the tip and edges. I have, however, generally found that if the patient takes biscuits and other dry food, which entail chewing and promote the flow of saliva, the tongue remains clean and moist throughout the illness, just as it does under similar conditions in typhoid fever. The slight diarrhœa, which occurs in more than half of the cases at the onset, is not severe, a small number of loose stools being passed for a few days, after which constipation is almost always present.

The abdomen may be normal in appearance, but it is more often full as in typhoid fever. There is generally no tenderness apart from that of the spleen. The spleen is palpable in at least two-thirds of the cases, and in my experience is found to be enlarged by percussion in all of the others. The enlargement is felt curiously far forward under the left rectus muscle, even when it is not felt further to the left. It is often much firmer than in typhoid fever

and may remain palpable for some days after the temperature has fallen to normal. It is generally tender in the early stages and may give rise to spontaneous pain, which is occasionally so severe that the onset of pleurisy is suspected. Even when it cannot be felt, pain is produced by deep palpation under the outer part of the left costal margin. The liver is not enlarged; the gall-bladder is occasionally tender owing to the presence of cholecystitis, which may give rise to no other symptoms.

In addition to the headache and abdominal discomfort, the patient often complains of pains all over the body, especially in the back and limbs and sometimes in the joints, which are, however, never swollen. Slight bronchitis is often present and may give rise to a troublesome cough. Though at first the patient generally has the heavy, inert appearance so often seen in typhoid fever, in most cases he looks and feels comparatively well by the second week, and he almost always feels still better at the end of the third week, even if the temperature is still raised. Only in a small proportion of cases does the toxic condition with mental dulness, characteristic of severe typhoid fever, develop.

In 75 per cent. of cases spots appear between the sixth and twelfth days. They come in crops, which last three or four days and are sometimes still visible after the temperature is normal. They are often larger, more irregular in shape, more raised and of a deeper red colour than in typhoid fever, and are sometimes remarkably profuse. They occur most frequently over the lower ribs in front, on the flanks, and on the back of the shoulders, and are often arranged in groups.

The temperature rapidly rises at the onset, usually reaching its maximum in 48 hours. The morning and evening chart is of a typically spiky character with daily variations of at least two degrees; it is generally remittent, ranging between 99° and 102° during the second week, but it may be intermittent. Continued fever is very unusual. In many cases the temperature does not reach 103°, and it is rarely higher than 104°. The duration of fever varies between one and eight weeks; in about half of the cases it is twenty days or less (Charts VII. and VIII.). Defervescence generally occurs rapidly by lysis, being often complete in two days. It may be followed by a sudden rise of temperature lasting for one to three days, and this is occasionally repeated

once or twice. One, and occasionally two, three, or even four true relapses, lasting six to fifteen days, with a return of pyrexia and often with splenic enlargement and a new crop of spots, occur in about 10 per cent. of cases after between two and eighteen days,

CHART VII.

Short case of paratyphoid fever A.

but most frequently after eight to ten days of apyrexia (Chart IX.). Rigors may be repeated throughout the illness in addition to the initial shivering attacks.

A slow pulse is very characteristic of paratyphoid fever, a

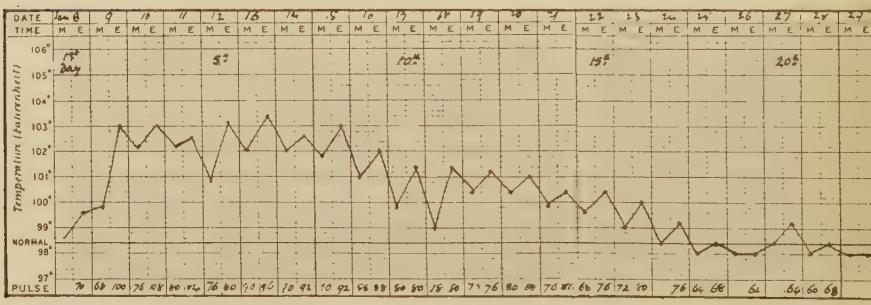


CHART VIII.

Paratyphoid fever B.

rate of 70 with a temperature of 102° being common; at the onset, however, the pulse is often quite rapid. The rate sometimes varies considerably without obvious reason, and its variations are independent of variations in temperature. The pulse is very soft

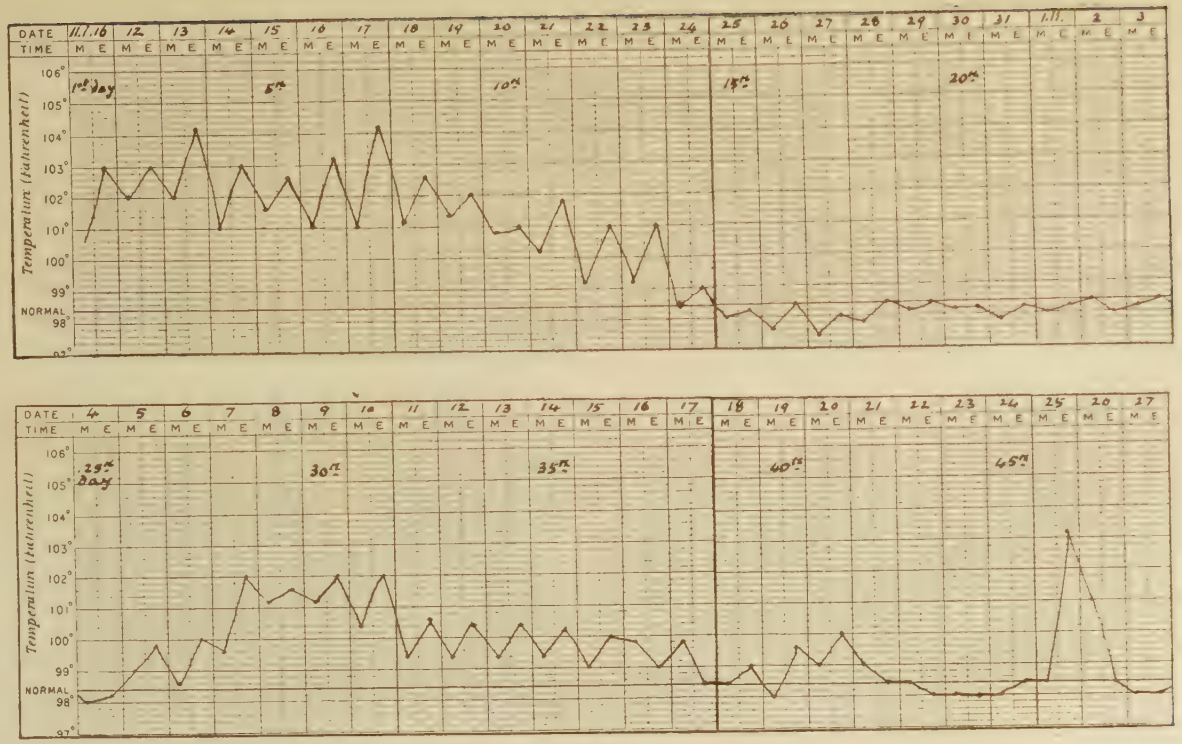


CHART IX.

Paratyphoid A with one long relapse and one short rise of temperature.

and often dicrotic, the blood pressure varying between 80 and 100 mm. of mercury. Bradycardia may persist after convalescence, but it gives rise to no symptoms. In a number of cases the heart is slightly dilated with a systolic murmur at the apex; slight cyanosis and coldness of the extremities are then observed, and tachycardia may be present and may persist for a considerable period, greatly delaying convalescence. If a patient with this condition returns to duty before the poisoned myocardium has completely recovered, he is very apt to develop a "soldier's heart" (*vide* p. 126).

Hæmorrhage occurs in paratyphoid B with the same frequency as in typhoid fever, but very rarely in paratyphoid A; it may be severe and even fatal. Perforation, which had never been observed in paratyphoid fever before the war, occurs in about 1 per cent. of cases both of paratyphoid A and B; the ileum, appendix, and colon are involved with approximately equal frequency. Meteorism is very rare and only occurs in the most severe and toxic cases.

Capillary bronchitis and broncho-pneumonia are comparatively common, especially in bad weather. The latter is a serious complication and in my experience is more frequent in paratyphoid A than B; it may develop at the onset, so that the case is at first regarded as simple pneumonia. In rare cases the broncho-pneumonia is complicated by a pulmonary abscess; the *B. paratyphosus* has been isolated from the sputum in pure culture. Pleurisy, empyema, infective endocarditis and pericarditis have been observed in rare cases. Thrombosis may occur in the femoral and saphenous veins; cerebral thrombosis probably explained the occurrence of convulsions accompanied by a rise in temperature for two days during convalescence in one case I saw, and also the transient hemiplegia which was reported in another case. Pharyngitis, tonsillitis and parotitis may occur; transitory deafness is not uncommon and is probably due to mild otitis media. In several of the Salonica cases pain was felt in the sole of the foot near the metatarso-phalangeal joints, and there was tenderness in this region, though the joints themselves were not affected; the pain began towards the end of the illness and generally lasted about a fortnight.

Slight albuminuria, which disappears at the defervescence, is

common: occasionally true nephritis with blood and casts in the urine is present, but œdema and other renal symptoms are never observed. The blood disappears in a few days and the albumin does not remain much longer. In a fatal case I saw at Salonica I found the kidneys large and congested with numerous minute abscesses and hæmorrhages under the capsule, but the renal pelvis was normal. As paratyphoid bacilli are often excreted in the urine, it is not surprising that pyelitis and in rare cases pyelonephritis may result. The bile is always found to contain the organism after death: this accounts for the occasional development of cholecystitis. Jaundice was a not uncommon complication in Gallipoli, where epidemic jaundice was also very prevalent: its rarity in France and Salonica, where jaundice was much less common, makes it probable that the association was generally accidental. Suppurative pylephlebitis is a very rare complication.

Suppurative orchitis, splenic abscess, periostitis and suppurative peritonitis without perforation have been observed, and I have also seen an abscess of the thyroid gland and an ischio-rectal abscess; in some cases the *B. paratyphosus* has been isolated from the pus.

Bacteriological Diagnosis.—The only absolute proof that paratyphoid fever A or B is present is the discovery in the blood, fæces, or urine of the bacillus with its characteristic cultural properties and its property of agglutinating with the specific immune serum, obtained from an animal immunised with the organism in question. In a few cases a bacillus has been isolated with all the cultural properties of *B. paratyphosus*, which does not agglutinate with the specific immune serum of either A or B; these cases are the result of infection with one or more allied organisms, which might be called *B. paratyphosus C* or *D*. As the bacillæmia is of short duration, beginning about the third day and rarely lasting more than a week, the blood should be taken for examination as soon after the third day as possible, and preferably in the evening when the temperature is generally at its highest. If a relapse occurs in a case in which the organism has not been isolated, hæmoculture should be tried again, as it may then be successful. The *B. paratyphosus* can often be found in the fæces, especially in the early stages when slight diarrhœa is so

common; when hæmoculture is negative, an attempt should therefore be made to isolate the organism from the fæces, an aperient being given if constipation is present, and if this fails from the urine.

In all cases coming under observation after the sixth day, and in cases seen earlier, but in which the organism has not been isolated from the blood, the agglutinating reaction of the serum should be tested against *B. typhosus* and *B. paratyphosus A* and *B* (Widal's reaction). If agglutination occurs with a high dilution with one of the paratyphoid organisms and either not at all or very slightly with the other and with the *B. typhosus*, no further examination is required; this will still be true when universal inoculation against paratyphoid A and B as well as typhoid fever is adopted. Occasionally agglutination takes place in considerable dilutions, but not in slighter dilutions; thus in a case of paratyphoid B Elworthy did not obtain agglutination until a dilution of 1 in 160 was used; if only weaker dilutions had been tested the reaction would thus have been missed. In men who have been inoculated against typhoid fever agglutination of *B. typhosus* but of neither paratyphoid organism is only important if it is obtained in a dilution of at least 1 in 100. If agglutination is absent or only occurs with feeble dilutions for one of the three organisms, or if agglutination occurs with more than one, one or more additional examinations must be made at intervals of five or six days. Infection is proved if there is a rise in agglutinating power with one of these organisms, whilst agglutination of the others is absent throughout or remains constant or diminishes. Absence of agglutination up to the twentieth day, or slight agglutination, which remains unaltered or diminishes and is therefore probably due to previous inoculation, is evidence against infection. Elworthy found, however, that agglutination occasionally disappears and subsequently reappears in a greater dilution than before. Positive agglutination results are of more value than negative results, as in rare instances agglutination never occurs, although the clinical evidence is almost conclusively in favour of a diagnosis of paratyphoid fever; this occurs particularly in very mild cases and in very severe cases in which there is no reaction. The test should be repeated in such cases just before the patient leaves hospital, as it occasionally becomes

positive even in late convalescence: thus Elworthy did not obtain a positive Widal reaction in a mild case of paratyphoid A until fourteen days after the temperature had fallen to normal. According to Dreyer and Walker the maximum agglutination titre of typhoid and paratyphoid infection occurs between the sixteenth and twenty-fourth day both in inoculated and uninoculated individuals. In some cases the reaction only becomes positive after a relapse.

Leucopenia is common, but is less constant than in typhoid fever, especially in the early stages and in mild cases; thus between 8000 and 14,000 leucocytes were present per c.mm. in several cases examined by Elworthy in Salonica. Complications such as broncho-pneumonia give rise to definite leucocytosis. A relative increase in lymphocytes is present in a larger proportion of cases; Elworthy rarely found less than 36 per cent., even when the total count was as high as 14,000. Such a differential count is not of course specific, as it is quite compatible with various other infections.

Differential Diagnosis.—Paratyphoid fever differs chiefly from typhoid fever in being a much milder infection with a much smaller mortality, but these differences are not present in patients who have received anti-typhoid inoculation. The profound toxic state, which is comparatively common in unmodified typhoid fever, s only seen in exceptional cases. Whereas in typhoid fever the patient tends to become more and more toxic as the disease progresses, in paratyphoid fever he is generally at his worst at the end of the first week, and even in severe cases, which give rise to the greatest anxiety in the early stages, remarkable improvement occurs before the temperature falls, and convalescence is unexpectedly rapid. The onset is much less acute, and the temperature rises more rapidly, remains raised for a shorter period and shows greater daily variations in paratyphoid fever, and there is rarely any period of continued high fever, such as is seen in the second week of ordinary typhoid fever. Shivering at the onset is much more common in paratyphoid than in typhoid fever, and rigors in the course of the illness are not so rare. The spleen is much firmer and is generally more tender, and the spots are often larger, more irregular in shape, and more raised. Lastly,

paratyphoid fever is very much more common than typhoid fever in individuals, who have been inoculated against the latter but not the former.

Many cases are at first diagnosed as *influenza*, especially when the onset is acute, and for a few days it is often quite impossible to distinguish between the two diseases. True influenza has, however, been comparatively rare in the present war. Apart from the bacteriological evidence, leucopenia with relative lymphocytosis points to paratyphoid fever, though its absence does not exclude the diagnosis ; the spleen is always either palpable or is found to be enlarged on percussion, but this is sometimes also the case in influenza. The temperature generally falls before the end of the first week in influenza, and the diagnosis is often settled about this time by the appearance of spots.

The onset of both forms of *trench fever* may simulate that of paratyphoid fever, but the course of the illness and the severe pain in the shins quickly indicate the nature of the infection. The temperature chart in prolonged cases of paratyphoid fever may resemble that of *Malta fever ;* in doubtful cases the diagnosis can only be settled by blood culture and agglutination reactions.

French observers have drawn attention to a group of cases, which are clinically indistinguishable from paratyphoid or typhoid fever, but in which the *Micrococcus tetragenus*, a Gram-positive diplococcus, or the *Bacillus coli* has been isolated from the blood in pure culture. In other cases the *B. typhosus* or *paratyphosus* has been found at the same time or at a preceding or subsequent examination, or a positive Widal reaction for the *B. typhosus* or *paratyphosus* has been obtained, although one of the former organisms was also isolated from the blood. It is probable, therefore, that these infections are in many cases secondary, the organisms perhaps being absorbed into the blood-stream from the intestinal ulcers, but this is certainly not always the case, so that the possibility of a tetragenus, diplococcus, or *B. coli* infection should be remembered.

Exceptional cases of paratyphoid fever, in which the onset is very acute with diarrhœa, vomiting and abdominal pain, may closely simulate *food poisoning ;* the course of the disease, the presence of spots, the enlargement of the spleen and the bacteriological examination will settle the diagnosis. When abdominal

pain and vomiting occur at the onset, especially if there is tenderness in the right iliac fossa, *appendicitis* may be simulated, but instead of the leucopenia with a relative increase of mononuclear cells seen in paratyphoid fever, leucocytosis with a relative increase of polymorphonuclear cells is present in appendicitis, and in most of these paratyphoid cases the suspicious symptoms rapidly disappear, though the temperature remains raised. As the appendix is often involved in the inflammation and ulceration of the bowel, it may give rise to peritonitis both with and without perforation. The early joint pains are in rare cases so severe that *rheumatic fever* is suspected; the joints are, however, never swollen, and the temperature does not at once fall to normal with salicylates. In exceptional cases *dysentery*, *cholera*, or *meningitis* may be so closely simulated that the possibility of paratyphoid fever is not considered, until the bacteriological examination of the stools and cerebro-spinal fluid demonstrate the absence of the specific organisms of these diseases and the presence of the *B. paratyphosus*. The possibility of a double infection with dysentery and paratyphoid fever must also be remembered, as this association was not uncommon in Gallipoli.

The æstivo-autumnal form of *malaria* may closely resemble paratyphoid fever; the differentiation can then only be made by a blood examination, as remissions may be slight, and typical rigors, though not uncommon in paratyphoid fever, do not always occur in malaria.

It is impossible to distinguish clinically between paratyphoid A and B, and the majority of the features which have at different times been said to be characteristic of one or other condition have not proved to be so on further investigation. This is particularly noticeable when the descriptions of the disease as seen in different areas of the war are compared; I have found that none of the symptoms, which have been said by observers in France to aid in the diagnosis, have been of any value in Lemnos or Salonica. It has been repeatedly stated that paratyphoid A is a much milder disease than paratyphoid B, but this has not been my experience, as many of the most severe cases as well as some of the least severe have been examples of paratyphoid A. The only symptom which has seemed to me to be generally distinctive is the very large and very hard spleen sometimes seen in paratyphoid A.

Prognosis.—The prognosis in paratyphoid fever is very much better than that in typhoid fever unmodified by previous inoculation. The mortality among 2118 bacteriologically proved cases in France was only 1·32 per cent., but it was probably rather higher in the Mediterranean Expeditionary Force, though not at Salonica. The chief causes of death are perforation, broncho-pneumonia, profuse hæmorrhage and toxæmia, which is also an important factor in many cases of death occurring from other causes. So far as my experience goes, and it agrees with the post-mortem records of Dawson and Whittington, the extreme end of the ileum was most severely ulcerated in cases in which toxæmia was the main or sole cause of death. The ileo-cæcal aperture was so much involved that there appeared to be some obstruction due to swelling of the mucous membrane, and to this might be added spasm or absence of the normal periodic relaxation of the ileo-cæcal sphincter, such as occurs in acute appendicitis. This would result in severe ileal stasis and consequent intestinal intoxication, which would aggravate the toxæmia due to the poisons produced by the *B. paratyphosus*. Death has also resulted from abscess of the spleen, gangrenous cholecystitis, and peritonitis without perforation. I performed the autopsy on a case of paratyphoid A at Salonica, in which death occurred from suppurative nephritis in the fifth week, when all the ulcers in the intestine had healed; the organism was isolated from the bile obtained from the gall-bladder after death.

The proportion of severely toxic cases is small, and ambulatory cases lasting only a few days with mild pyrexia, slight headache, splenic enlargement and perhaps spots occur, though their frequency is not known, as the majority doubtless escape recognition. Even in the worst cases convalescence is comparatively rapid.

The faster the pulse in relation to the temperature the more severe is the infection, a rate of over 100 after the fifth day indicating a case of some severity. Severe bronchitis, much mental clouding, and abdominal distention are the most serious symptoms. The prognosis is much better in warm than in cold weather, when the disease is very liable to be complicated by broncho-pneumonia and relapses are more common. In one area occupied by the French Army the mortality was 6·25 per cent. in February, 1915, but almost zero the following spring and summer.

Prophylaxis.—The prophylaxis of paratyphoid fever consists in recognising and isolating cases as early as possible, preventing contamination of food and water with infective material from their excreta, and immunising all who may be exposed to infection by anti-paratyphoid inoculation. All suspicious cases should be sent into hospital without delay; familiarity with the clinical features of the disease will lead those in charge to call in the aid of a bacteriologist at an early stage, when diagnosis by blood culture is possible. As soon as the diagnosis is made, the man's unit should be informed, so that his bedding may be disinfected and a search made for the source of infection. There is no real need to isolate cases in special hospital wards, though it may be convenient to do so, especially if inexperienced orderlies are in charge. The fæces and urine should be disinfected immediately and incinerated as quickly as possible; as the sputum may also be infective, it should be disinfected and patients should not be allowed to cough into the air but into paper handkerchiefs, which can be burnt. It is advisable to isolate convalescent patients until their stools and urine are proved to contain no paratyphoid bacilli on three consecutive occasions at intervals of a week, in order to avoid sending carriers back to their units. If it were not for this, a man who has had paratyphoid fever would often be fit for light duty a month after his temperature has fallen to normal. Mild cases, which have escaped recognition, are a far greater danger as a source of infection than convalescent carriers. For this reason the urine and fæces of all soldiers should be dealt with as described in the chapter on dysentery, and the same precautions should be taken with regard to the protection of food from flies and dust.

The rarity of typhoid fever compared with paratyphoid fever in the army is the best proof of the efficacy of anti-typhoid inoculation. Universal inoculation against the paratyphoid fevers would result in the almost complete disappearance of the disease; if a mixed typhoid and paratyphoid vaccine had been used from the beginning, there would probably have been only twenty cases of paratyphoid fever among the British troops in France during the first sixteen months of the war, or about 5 per cent. of the number of cases of typhoid fever, as this appears to be the relative frequency of the fevers in the uninoculated civil population. All men who have not been inoculated against typhoid fever

within twelve months should receive a combined vaccine of 500 millions *B. typhosus*, and 250 millions each of *B. paratyphosus A* and *B;* otherwise a mixed paratyphoid vaccine should be used. The dose is repeated in a week.

Treatment.—Ever since a visit to America ten years ago, when I saw the remarkably good effect of a generous diet in shortening convalescence and in reducing the liability to septic complications without increasing the danger of hæmorrhage or perforation, I have treated all typhoid patients in this way; I have never had occasion to regret it, nor have I ever heard of any ill-result. It is the duty of those in charge of sick soldiers to make them fit for active service as quickly as possible, and I am quite certain that a diet consisting of milk puddings, custard, eggs, and bread and butter in addition to milk makes a man fit for duty very much sooner than he would be on a semi-starvation diet of milk alone. Such articles of diet are completely fluid by the time the ulcerated area of the ileum and colon is reached; on the other hand, I have several times seen clots in these parts of the bowel after the death of patients who had been on a purely milk diet. Biscuits, which the patient has to chew, are of the greatest value, as they stimulate the secretion of saliva and keep the tongue moist. I have never seen the so-called typhoid tongue in a patient treated in this way; it is simply a result of a diet which requires no chewing and which calls forth no secretion of saliva. Complications such as parotitis are directly due to a septic condition of the mouth, which can easily be prevented by good nursing if a suitable diet is given. An abundant supply of fluid should be given, and in severe toxic cases normal saline solution should be administered per rectum by the drop method, or in urgent cases by subcutaneous or intravenous injection.

The only drug which is probably of any use is hexamine (urotropine). It is a powerful urinary antiseptic and is probably a biliary antiseptic as well. As the chief source of the organism in carriers is the gall-bladder, and the second most important source is the urinary tract, the number of convalescent carriers would be very greatly reduced if it were possible to keep these parts sterile by the systematic use of hexamine. The drug should be given in doses of gr. x three times a day until the patient leaves

hospital. It has the additional advantage of diminishing the liability to cholecystitis and pyelitis.

The patient should not be allowed to get up even in the mildest cases until his temperature has been normal for a week. I have seen fatal perforation occur in a case of paratyphoid A just when defervescence appeared to be almost complete, and at the autopsy numerous ulcers, only a few of which showed signs of healing, were still present, so that a fall in temperature does not always indicate that the ulcers have healed. When the heart is dilated, prolonged rest in bed is necessary, and smoking should not be allowed during convalescence.

I am particularly indebted to Lieut. R. R. Elworthy, Pathologist to the 29th General Hospital at Salonica, for help in the description of the laboratory methods of diagnosis, which proved exceptionally successful in his hands, and to Major C. H. Benham and Lieut. W. H. Fleetwood of the same hospital for some of the clinical data.

References

Proceedings of the Royal Society of Medicine, Medical Section, IX. 1, 1915. (Discussion on Paratyphoid fever.)

J. A. Torrens and T. H. Whittington: *British Medical Journal*, 1915, II. 697. (Paratyphoid in France.)

W. H. Willcox: *Lancet*, 1916, I. 454. (Paratyphoid Fever in the Mediterranean Forces.)

J. Carles: *Les Fièvres Paratyphoides*, Paris, 1916. (Paratyphoid Fever in the French Army.)

Sir B. Dawson and T. H. Whittington: *Quarterly Journal of Medicine*, IX. 98, 1916. (Study of Fatal Cases.)

C. S. Martin and W. G. D. Upjohn: *British Medical Journal*, 1916, II. 313.

G. Dreyer and E. W. Ainley Walker: *Lancet*, 1916, II. 419.

CHAPTER VI

EPIDEMIC JAUNDICE

(*a*) *Bacillary Jaundice*

Though the symptoms of the epidemic jaundice, which occurred in the Mediterranean Expeditionary Force, are similar to those of the catarrhal jaundice, which occurs sporadically among civilians in peace time and is not uncommon among soldiers in the field, the disease is clearly a distinct one, as shown by its epidemic nature, the frequent presence of symptoms pointing to a general infection or toxæmia, such as enlargement of the spleen and nervous symptoms, and the results of the bacteriological investigations carried out at Gallipoli.

Epidemiology.—Over 70,000 soldiers were affected with jaundice in the American Civil War, and in the South African War 5648 cases occurred among the British troops. In the present war the first outbreak of epidemic jaundice occurred in July, 1915, among the troops stationed in certain camps in Egypt. There was no jaundice at Gallipoli until the middle of August, when it suddenly appeared in a large number of different units in all parts of the Peninsula; it then rapidly spread among the men of each unit. Six weeks later cases began to arise in Lemnos and Imbros. This is most easily explained on the assumption that the infection was conveyed to different parts of the Peninsula by convalescent or contact carriers in drafts from the infected Egyptian camps, and that the subsequent spread to the islands was caused by patients coming from Gallipoli. The epidemic reached its greatest intensity in the second half of October. Official statistics give no adequate idea of the prevalence of the disease, as large numbers of men continued at duty throughout their illness, especially in November and December; several regimental medical officers told me that at times as many as one-tenth of their men actually

in the trenches were jaundiced. In one battalion there were a hundred cases during October, but only thirty-six were regarded as of sufficient severity to be sent into a Field Ambulance.

Epidemic jaundice was as frequent among the French as the British at Gallipoli, but intelligent prisoners stated that no cases occurred among the Turks, although their first line was situated only a few yards from that of the Allies, and their sanitary arrangements were infinitely less good.

Epidemic jaundice occurred, though much less frequently, among the British and French troops, who went from Gallipoli to Serbia in October, 1915, and a few cases continued to develop among them after their return to Salonica until the beginning of 1916, when the epidemic ceased. Only a few sporadic cases of catarrhal jaundice occurred among the British troops at Salonica who had come from France or England, and there were no cases among the French who came direct from France.

Pathology.—No organisms were isolated from the blood in cases of epidemic jaundice occurring among British troops, except occasionally when it was complicated by the presence of ordinary paratyphoid fever. This is probably due to the fact that the cases were only examined after the jaundice had actually appeared; at this stage the general symptoms and fever are less marked than in the pre-icteric period, and any bacillæmia which may have been present has probably disappeared. Two French observers, Sarrailhé and Clunet, who worked in an underground laboratory in a Turkish village on the Peninsula itself, obtained blood from large numbers of patients with fever, anorexia and abdominal discomfort, which often proved to be the symptoms preceding the onset of jaundice. In 112 cases—about half of the number examined—they succeeded in cultivating an atypical paratyphoid organism, which differed slightly from that of paratyphoid A and B in its cultural characters and did not agglutinate with the specific immune sera of A or B, but agglutinated with the serum obtained from the same patient or from other patients suffering from the disease. They suggested calling the organism *B. paratyphosus Dardanellensis*. In a later publication, however, they state that the cultural properties and agglutination reactions of the 94 cultures they brought home from the Dardanelles had altered

when examined six months later; 51 were now typical and 33 atypical paratyphoid A, 5 typical and 1 atypical paratyphoid B, and 4 atypical typhoid. The conditions present at Gallipoli had thus resulted in the variation of the properties of the paratyphoid and typhoid bacilli, associated with a change in the pathological condition they produced. But the variation was too transitory to justify the classification of the Dardanelles organism as a distinct species, and the name *B. paratyphosus Dardanellensis* should therefore be given up.

The normal duodenal contents are almost sterile, strongly alkaline, bile-stained and clear when first removed, though they become cloudy on standing. I aspirated the contents of the duodenum by means of Einhorn's evacuator * in nine cases of epidemic jaundice at Lemnos. The fluid was neutral, very slightly alkaline, or even slightly acid; it was generally turbid and contained excess of mucus and large numbers of bacteria, but no paratyphoid-like bacilli; its colour was often, though not invariably paler than normal, but bile was always present in it. The gastric contents were normal in appearance, and contained few or no bacteria.

In the only uncomplicated fatal case, in which a post-mortem was performed at Lemnos, catarrh of the duodenum, bile ducts and gall-bladder was found by Major C. J. Martin. A similar condition was found in cases of jaundice complicating paratyphoid fever. It is still uncertain whether the primary infection occurs in the duodenum, from which it spreads up the bile ducts and causes catarrhal cholangitis and cholecystitis, which always subside without suppuration, or whether a primary infection of the blood is followed by excretion of the organism in the bile, by means of which the ducts and the duodenum are secondarily infected. The onset of the disease with digestive symptoms before jaundice appears and before the liver and gall-bladder become enlarged and tender point to the former view as the more probable, the disease being due to a specific infection with a paratyphoid-like organism, which differs from the *B. paratyphosus A* and *B*

* This is a small perforated metal bulb attached to a Southey's tube. The bulb is swallowed with some milk; after about 2½ hours it passes into the duodenum, the contents of which are aspirated. The tube is then partially withdrawn in order to obtain the gastric contents.

in attacking the duodenum instead of the ileum and colon. The swelling of the mucous membrane of the duodenum at the mouth of the common bile duct gives rise to obstructive jaundice, but the infection may exist without any jaundice, the degree of which depends upon the anatomical relations of the ducts in each individual.

Etiology.—The only channel by which the organism can leave an infected individual is the fæces. Infection of other individuals must be due to food and water, which have become contaminated by the fæces of patients suffering from the disease, but dust and flies are less important in this connection than in dysentery and paratyphoid fever, as the disease was most prevalent in the autumn and early winter when there were no flies and often no dust.

There is no doubt that exposure to cold and fatigue predispose to epidemic jaundice by lowering the individual's resistance, and this is probably one reason why the disease became so prevalent at Gallipoli in the last months of 1915. But these factors are not always present, as the epidemic in the Egyptian camps occurred in very hot weather, and only five out of fifty consecutive cases at Lemnos, in which special inquiry was made on this point at the end of November and beginning of December, ascribed their illness to anything of the kind.

In view of the extreme frequency of diarrhœa at Gallipoli it is noteworthy that epidemic jaundice developed most commonly in men whose bowels had been regular for some time before the onset of symptoms, although two-thirds had suffered from diarrhœa, often on several occasions, since their arrival on the Peninsula. Indeed constipation was noted twice as often as diarrhœa as the immediate precursor of the disease.

Jaundice only became common when the incidence of dysentery was on the decline, and the occasional association was no greater than could be explained by the great frequency of both diseases. Moreover, in the majority of cases an interval, which was often considerable, occurred between the disappearance of all dysenteric symptoms and the development of the jaundice ; in the remaining cases the onset was more or less simultaneous, or the jaundice developed with its typical premonitory symptoms at any stage

during the patient's residence in hospital. The association was therefore of the same accidental nature as that with wounds, several cases of jaundice having developed in patients who were in a Lemnos hospital, in a Hospital Ship on the way to England, or even in English hospitals on account of wounds received on the Peninsula.

The association of paratyphoid fever was undoubtedly accidental when a considerable interval elapsed between the onset of the two diseases, but the discovery of the *B. paratyphosus A* or *B* in the blood during life and in the gall-bladder after death, when jaundice developed in the course of an attack of paratyphoid fever, led some observers to believe that the jaundice was then due to the paratyphoid infection, and even that the cases, in which no symptoms of paratyphoid fever were found and the organism was not isolated from the blood, were also due to a mild infection with the *B. paratyphosus A* or *B*. But the *B. paratyphosus* is constantly found in the gall-bladder of fatal cases of paratyphoid fever whether jaundice is present or not, so that its discovery in fatal cases complicated by jaundice is no proof, as has been too readily assumed, that the jaundice is due to the paratyphoid fever and is not an accidental complication. The extreme rarity of jaundice in typhoid fever in spite of the constant infection of the bile with the *B. typhosus*, and its absence as a complication of paratyphoid fever in France and Salonica render it very probable that the association is almost always accidental; moreover the frequency of jaundice in paratyphoid fever at Lemnos was no greater than its frequency among patients admitted for other conditions.

Chronic indigestion sometimes preceded the onset of jaundice, but this again was no more common than would be expected from the frequency of indigestion due to indigestible food and bad teeth.

Epidemic jaundice has thus no direct causal relationship with diarrhœa, dysentery, paratyphoid fever, or chronic indigestion, though these diseases may predispose to infection by lowering the general resistance of the individual.

Symptoms.—In all but about five per cent. of cases the jaundice was preceded by other symptoms, the most frequent interval between the onset and the development of jaundice of

sufficient intensity to be noticed by the patient or his companions being three days, two days being the next most common interval; in several cases it was between four and seven days, but it was rarely less than two or more than seven. When a longer interval than seven days was recorded, the onset was generally obscured by chronic indigestion which had already been present for a fortnight or more.

Pre-icteric Symptoms.—The most common early symptom is anorexia, which may develop gradually in the course of a couple of days, but is often noticed quite suddenly at one meal, generally breakfast, the previous meal having been enjoyed as usual. The anorexia quickly becomes extreme for all solid food, but the patient is generally ready to drink milk, though occasionally he may for a short time be unable to take even fluids. The mere sight of food makes him feel sick, and if he attempts to eat he is likely to vomit. The anorexia is soon followed and in a few cases is immediately preceded by a feeling of epigastric discomfort, which may develop into actual pain, which is constantly present, but is increased directly any solid food is taken. In only two cases out of a consecutive series of fifty was anorexia absent either at the onset or during the course of the illness. Contrary to what might be expected, the tongue remains clean.

Headache of moderate severity is often present, and may be accompanied by a feeling of swimming in the head and giddiness in the erect position, which is sometimes so great that the patient is hardly able to stand or walk. In many cases he feels very weak and disinclined to exert himself. These symptoms occasionally precede or develop simultaneously with the gastric symptoms, but more commonly they follow a day or two later.

In the comparatively rare instances in which the temperature was taken before the onset of jaundice it was often found to be raised to 100°, and occasionally even to 103° or 104°, when the headache was unusually severe with considerable constitutional disturbance, the onset simulating that of influenza or paratyphoid fever.

Symptoms after the onset of Jaundice.—The patient occasionally notices that his urine is becoming dark, but more often the jaundice is first noticed by his friends, or by the medical officer

when he reports sick on account of the premonitory symptoms. The jaundice is never very intense and may be so slight that it is not recognisable except in the conjunctivæ. Its intensity is no indication of the severity of the infection, as pronounced jaundice may occur with no other symptoms, so that one man may be able to continue at duty throughout his illness, whilst another may appear and feel very ill although the jaundice is quite slight. The infection of the bile ducts and duodenum does not necessarily produce sufficient obstruction to cause jaundice. Thus Captain Jolly saw several cases at Anzac, in which the symptoms made him expect that jaundice was about to develop; though bile appeared in the urine, no staining of the conjunctivæ or skin occurred. The jaundice reaches its greatest intensity in a few days and then slowly diminishes, a yellow tinge being often visible a fortnight after all other symptoms have disappeared.

The appetite begins to return a few days after the onset of jaundice. The epigastric pain is almost always accompanied by a moderate degree of tenderness; somewhat less severe pain is felt across the lower part of the back. Occasionally there is a vague pain in the region of the gall-bladder and liver, but never in the right shoulder. After a few days the pain disappears, slight epigastric discomfort remaining for a short time longer. The bowels tend to be constipated, and considerable relief is experienced when they have been sufficiently but not excessively opened. In exceptional cases slight diarrhœa is one of the premonitory symptoms, but it generally disappears soon after the patient becomes jaundiced. The fæces retain their normal colour in more than half of the cases; in the remainder they become pale and sometimes definitely clay-coloured a day or two after the first appearance of jaundice.

Tenderness is almost invariably most marked over the gall-bladder; deep pressure may cause little or no pain with shallow respiration, but on deep inspiration acute pain, which may radiate to the epigastrium, is felt the moment the gall-bladder comes into contact with the fingers. The gall-bladder was definitely palpable in about one-tenth of the cases. If every patient were examined regularly from the onset of the symptoms, it would probably be felt in a considerably larger proportion of cases, which would be still greater were it not for the tendency of the rectus

muscle to contract when palpation is attempted. In a few cases the gall-bladder remains enlarged when the tenderness has almost disappeared; it can then be easily palpated.

The liver is often slightly enlarged: owing to the rigidity of the abdominal muscles this may only be recognisable by percussion, but in about a third of the cases the lower border is palpable, generally about half an inch below the costal margin in the right nipple line. The liver is firm and at first moderately tender, but the enlargement may persist after all tenderness has disappeared, occasionally for as long as fourteen or twenty days. In a small proportion of cases the spleen is enlarged and slightly tender. This must be due to a general infection secondary to that of the alimentary canal, and it is consequently most marked in severe and febrile cases, especially in the pre-icteric stage.

Bile is always present in the urine. A small quantity of albumin is sometimes found, and occasionally a few casts.

As soon as the jaundice appears the pulse becomes slow; it is often only 60, but with the exception of one case in which it fell to 38 the day after the jaundice appeared, rising to 72 four days later, I have not seen a case in which it was below 52. The rate begins to increase again about the fifth day, and it is generally normal in ten to fourteen days. In about a quarter of the cases both sides of the heart are slightly dilated. The dilatation is occasionally considerable, especially to the right of the sternum; the pulse is then rapid and dicrotic and the blood pressure is low.

Even if the temperature is high in the pre-icteric period, it falls with the onset of the jaundice, and either becomes normal at once or remains between 99° and 100° for three or four days. A higher or more prolonged pyrexia indicates that some other infection is also present.

The headache and vertigo observed in the pre-icteric stage generally disappear very soon after the onset of the jaundice. The patient becomes extremely weak in severe cases. Some medical officers were impressed by the frequency during convalescence of tremor of the hands, which was generally associated with tachycardia. In all cases I saw of this kind the patient had been unfit for some time before the onset of the symptoms which immediately preceded the jaundice; the absorption of bile salts and the rest in bed generally steadied the pulse, which became more

rapid when he got up. The picture of this post-icteric tremor and tachycardia was identical with the picture seen in many exhausted soldiers from the Peninsula, who had had no jaundice, and I regard the association as purely accidental.

The severe form of peripheral neuritis, clinically indistinguishable from beri-beri, which was an occasional sequel of epidemic jaundice, is described in the next chapter. Pruritus was never observed, probably owing to the comparatively short duration of the jaundice.

In the Egyptian epidemic and in the earlier Gallipoli cases the dilatation of the heart was more constant and more considerable than in the cases which occurred at Gallipoli and Lemnos in November and December. The initial pyrexia was higher and the enlargement of the liver was more common and more considerable. In Egypt the spleen was always considerably enlarged; it was often very tender and gave rise to spontaneous pain. This symptom was rarely so marked in Gallipoli, even in the earlier cases.

Prognosis.—Even among the severe cases in the Egyptian epidemic the prognosis was good with regard to life, no deaths having been recorded. I only heard of two Gallipoli cases, in which death occurred in the absence of some intercurrent disease, such as paratyphoid fever. In one the patient died from heart failure when convalescent from the jaundice, extreme dilatation of the heart being found post-mortem; in the other, recorded by Colonel W. H. Willcox, death occurred from toxæmia, which gave rise to delirium, vomiting and finally coma.

In cases of moderate severity the patient is generally fit for duty between three and six weeks after the onset of symptoms, if he was previously in good health. If the patient's health was already impaired at the time of infection, as was often the case at Gallipoli, the added toxæmia is likely to weaken him to such an extent that he will require two or three months to recuperate.

The chief causes of prolonged convalescence are general asthenia and dilatation of the heart, with a tendency to dyspnœa, vertigo, and tachycardia on exertion. A man should not be sent back to duty whilst his heart is still dilated, as otherwise he is very likely to develop a "soldier's heart" (*vide* p. 126).

Prophylaxis.—Although epidemic jaundice is not in itself a serious disease, as it is hardly ever fatal and does not necessarily prevent a man continuing at duty, it is liable to become so widely prevalent that it may form a very serious source of invaliding.

Owing to the similarity of their etiology the precautions which are necessary for the prevention of typhoid and paratyphoid fever are also applicable to epidemic jaundice. All cases should be promptly notified and sent to hospital immediately the diagnosis is made. Sisters and orderlies should be instructed to regard cases of epidemic jaundice as infective, and should take the same precautions to prevent the spread of infection and to prevent contracting the disease themselves as in typhoid and paratyphoid fever.

Treatment.—The patient should be kept warm in bed on a fluid diet until his appetite comes back and the abdominal tenderness disappears. He should then rapidly return to a full diet. A dose of calomel or castor oil should be given at the onset, but this should not be repeated unless constipation is present. It would be reasonable to give sodium salicylate (gr.20 three or four times a day) in order to disinfect the biliary passages, and perhaps in this way to prevent the patient becoming a convalescent carrier of the infection, but the drug does not seem to influence the course of the disease.

References

E. B. Gunson and J. W. C. Gunn: *Lancet*, 1915, II. 1294.
W. H. Willcox: *British Medical Journal*, 1916, I. 297.
A. Sarrailhé and J. Clunet: *Bull. de la Soc. Méd. des Hôpitaux de Paris*, XL. 45 and 563, 1916.

(*b*) *Spirochætal Jaundice*

In the spring and summer of 1916 a small epidemic of acute infective jaundice occurred among British troops in France. It differed in etiology and symptoms from the epidemic jaundice of Gallipoli, but closely resembled the disease, which occurs in epidemic form in Japan and was formerly common in Alexandria and Smyrna, but has not attacked British troops in the East during the present war. It has been referred to as Weil's disease, but this name should be discarded, as a similar condition was accurately

described by various French observers several years before Weil. The Japanese jaundice has recently been shown by Inada, Ido, and others to be due to infection with a spirochæte, the *Spirochæta icterohæmorrhagica*, which is present in the blood during the first week and the urine during the first four weeks. Animals develop jaundice on inoculation with the blood of a patient, and the organism can be recovered from their blood and viscera. Very soon after the results of the Japanese observations became known in France, a number of investigators succeeded in isolating the organism from the blood and urine of patients, and confirmed the animal experiments. From observations at two operations and three autopsies Sir Bertrand Dawson and W. E. Hume conclude that the jaundice is due to obstruction of the biliary papilla by œdema and congestion of the duodenal mucous membrane; the liver, gall-bladder, and bile ducts are normal, but the bile is unusually thick. The pancreas is healthy, and in spite of the changes in the urine no nephritis is present.

Symptoms.—The onset is most frequently gradual with general unfitness, headache, anorexia, nausea, and occasionally vomiting, but it may be sudden with shivering and great prostration. The temperature rises to between 102° and 104° and remains high for about a week; the fever often recurs a few days later. The tongue is dry and furred; the whole abdomen is somewhat painful and tender, and the patient passes constipated stools, which are merely clay coloured and occasionally contain bile. The liver is slightly enlarged and tender, but the spleen is generally normal; the lymphatic glands are often enlarged. The patient complains of lassitude and pains in the head, back, and limbs. The conjunctivæ are injected and herpes labialis is common. Jaundice appears between the second and seventh day: it does not vary with the degree of toxæmia, and in a few cases Dawson and Hume observed similar symptoms without jaundice in patients, whose blood and urine contained the spirochæte, and whose blood produced jaundice in guinea-pigs. The urine contains bile for four or five weeks; albumin is generally present with hyaline and granular casts, and sometimes red corpuscles. Hæmatemesis, hæmoptysis, epistaxis, and purpura are common in severe cases. The heart is not enlarged.

The disease thus differs from the Gallipoli epidemic jaundice in the more prolonged pyrexia and dry furred tongue, the tendency to hæmorrhages and conjunctival congestion, the character of the urine, and the absence of dilatation of the heart and enlargement of the gall-bladder and spleen.

Prognosis.—Only 6 out of the 91 cases seen by Stokes and Ryle and by Dawson and Hume died. The disease is thus milder than the form occurring in Japan and Alexandria, in which the mortality was about 30 per cent. Dawson and Hume classified 18 of their cases as severe and 58 as mild: in the former the illness lasted about three weeks and in the latter about one, but few patients were fit for duty in less than three months.

Treatment.—The urine should be disinfected for the first six weeks, as it is probably the chief cause of infection. Japanese observers conclude from their animal experiments that salvarsan is of little value, but the serum of immunised goats exerts a favourable influence on the disease.

References

R. Imada, Y. Ido and others: *Journ. Experim. Med.*, XXIII. 377, 1916.
N. B. Gwynn and J. J. Owen: *Lancet*, 1916, II. 518.
A. Stokes and J. A. Ryle: *British Medical Journal*, 1916, II. 413.
Sir B. Dawson and W. E. Hume: *Quarterly Journal of Medicine*, Oct., 1916.

CHAPTER VII

A FORM OF BERI-BERI OCCURRING AMONG BRITISH TROOPS

In October, November and December, 1915, a number of cases of peripheral neuritis associated with circulatory symptoms occurred at Lemnos among men invalided from Gallipoli, and at least one case occurred in a medical officer who had never been to the Peninsula. A few similar cases were subsequently observed by Major Shellshear in Egypt, and by Lieut.-Col. Willcox among soldiers invalided home from Mesopotamia. Owing to the striking resemblance of the symptoms in well-marked cases to those of beri-beri, the condition was at first regarded as ordinary beri-beri and due to the diet being deficient in certain essential constituents. It was soon noticed, however, that a considerable number of cases were associated with epidemic jaundice, and an examination of seventeen cases, which included all I was able to discover in Lemnos during December, led me to the conclusion that the latter infection was as important a factor as food deficiency, though this does not seem to have been the case in Mesopotamia.

Etiology.—A diet of pure proteins, carbohydrates, and fats with salts and water does not maintain health, unless a minute quantity of certain substances, known as vitamines, are added. The absence of one of these, a soluble non-protein nitrogenous substance, which contains no phosphorus and is destroyed at 130° C. but not at 100° C., is the chief cause of ordinary beri-beri. The absence of another, which is destroyed already at 70° C., causes scurvy. Beri-beri is most common among races whose staple diet is machine-polished rice, the husk and aleurone layer of which have been removed. Rice prepared in this way contains none of the vitamine required to prevent beri-beri, but this is present in sufficient quantity in the aleurone layer. The addition of an extract of rice-polishing or the substitution of rice, from which

the husk has been removed by steam or hot water which leaves some of the aleurone layer, prevents the development of beri-beri or cures it if it has developed. But beri-beri may also develop in individuals who eat no polished rice, if their diet contains an insufficient quantity of the necessary vitamine. As the heat used in the sterilisation of tinned food destroys the vitamine, a diet consisting mainly of tinned food with no fresh eggs, vegetables, or meat may cause beri-beri.

The facts which will presently be recorded show that it would be impossible to explain the outbreak of the disease among the British troops at Gallipoli solely as a result of a deficiency in the diet: it is necessary therefore to recall the observations on beri-beri made over ten years ago by Hamilton Wright, although they have recently been to a great extent ignored owing to the general acceptance of the food-deficiency theory. Hamilton Wright came to the conclusion that the disease was due to a specific infection of the duodenum, the toxins produced having a specific action on the nervous system. He was led to this view by the fact that acute and subacute cases begin with anorexia, especially for solid food, a dull heavy or burning sensation in the epigastrium, which is prominent and tender, diarrhœa and occasionally vomiting. The nervous symptoms appear between 5 and 72 hours later, but in a few cases simultaneously with the digestive symptoms. In cases which die in the early stages, the mucous membrane of the pyloric end of the stomach and duodenum is congested and shows minute hæmorrhages; microscopically the mucous membrane and submucous tissue were infiltrated with round cells and bacilli, and well-marked necrosis of the epithelium is present, leading to superficial sloughing of the mucous membrane. The duodenitis disappears in about three weeks, and is therefore not found in patients dying after the acute stage has passed, when nothing but residual paralysis and cardiac weakness are present. Similar pathological changes have been recorded by other observers, including Wernich, Anderson, Simmons and Miura.

Association with Jaundice.—In fourteen out of my seventeen cases (82·4 per cent.) the symptoms followed an attack of epidemic jaundice at intervals varying between three and thirty days from the onset of the jaundice, the average interval being eighteen days. In one case seen by Willcox the jaundice only began three weeks

after the onset of neuritis. Of the three cases in which there was no jaundice one patient had an attack of vomiting with epigastric pain and anorexia fifteen days before the onset of the neuritis; in both of the other cases the neuritis was immediately preceded by diarrhœa, the patient having had dysentery some time before. It has been suggested that the association was accidental and simply due to the fact that jaundice was very common on the Peninsula, but neuritis occurred at least eight times as frequently among patients who had had jaundice as it would have done if the association were simply accidental. In eleven cases of beri-beri in British troops from Mesopotamia Willcox only obtained a history of recent jaundice in one and of chronic diarrhœa in another, but in most of his cases both from Gallipoli and Mesopotamia epigastric discomfort and flatulence, generally associated with tenderness over the duodenum, were early symptoms.

The association of these cases of neuritis with epidemic jaundice is strikingly similar to the association of diphtheritic neuritis with diphtheria; the patient is generally fit in every way till the onset of the symptoms which immediately precedes the development of jaundice. His convalescence from jaundice appears to be normal, but within a few days of getting up after the attack is over the symptoms of neuritis appear.

It must be remembered that jaundice is merely a symptom and not necessarily an essential part of the disease, which is known for the sake of convenience as epidemic jaundice. In some cases severe jaundice occurs with very trivial symptoms, whereas in others the digestive and general disturbance is considerable, but the jaundice is very slight. Cases probably occur in which the duodenitis, which I believe was the primary condition in the Gallipoli epidemic jaundice, does not give rise to sufficient obstruction either at the mouth of the common bile-duct or by spread of the infection to the smaller ducts for jaundice to occur. This may well explain the occasional absence of preceding jaundice in these cases of peripheral neuritis. In a case already referred to symptoms occurred a fortnight before the onset of the neuritis, which were in every way similar to the prodromal symptoms of epidemic jaundice, and the constant anorexia and abdominal discomfort caused by the chronic diarrhœa in the two remaining cases might have prevented these prodromal symptoms from being recognised.

Such cases of neuritis following the infection but without jaundice would be strictly comparable to the cases of diphtheritic paralysis, in which the preceding diphtheritic pharyngitis escapes recognition.

Just as the preceding infective process may be latent owing to the absence of jaundice, so I believe may the neuritis be latent. Major Shelshear and I examined twenty consecutive convalescent patients, who had become jaundiced between three and twelve weeks before and were now in a convalescent depôt in Mudros West. In no less than eleven cases we found definite signs pointing to the presence of peripheral neuritis, generally associated with cardiac dilatation and tachycardia and in two cases with slight œdema of the ankles, and in three others we found some slight paræsthesia of the feet, tenderness of the calves or difficulty in rising from a squatting attitude, together with slight cardiac dilatation. Several of the patients showed some tremor of the hands or general unsteadiness, and others had a dilated heart with a rapid pulse, but we did not include these cases among the abnormal ones unless signs of neuritis were also present, as similar symptoms were not uncommonly found among patients convalescent from various other conditions and even among apparently healthy soldiers from Gallipoli. The patients had either not noticed anything abnormal themselves or had not thought the symptoms sufficiently important to mention. It is difficult to draw a line between these cases and the slighter ones of definite "beri-beri." If we are correct in regarding them as examples of latent neuritis following epidemic jaundice, it becomes all the more probable that the severe cases are directly due to the same infection as that which causes the jaundice. A similar conclusion may be drawn from the condition of the heart. It was recognised by all observers during the epidemic of jaundice at Gallipoli that the heart was very frequently dilated and the pulse sometimes very rapid after the initial bradycardia had disappeared. It is not surprising therefore that a similar dilatation should be observed in the cases of peripheral neuritis.

It has also been suggested that the association of beri-beri with jaundice might be due to the strict dieting given for the latter together with the associated toxæmia being sufficient to act as a final stimulus to the development of the disease, the

occurrence of which was already imminent owing to certain deficiencies in the diet. But if this were the case peripheral neuritis of the same character should have been still more common after dysentery and paratyphoid fever, as the former at any rate caused more invaliding than epidemic jaundice, and in both diseases the strict dieting was likely to be much more prolonged and the toxæmia much more serious. But I know of only two cases which followed dysentery and none which followed paratyphoid fever.

The earliest cases of peripheral neuritis were recognised about the middle of October, 1915, and they continued to occur in small numbers until the evacuation of Gallipoli; allowing for the interval which elapses between the onset of the jaundice and that of the neuritis, the incidence of the latter is what would be expected if it were due to the same infection, as the epidemic of jaundice assumed serious proportions about the middle of September. If the neuritis were solely due to deficiency of certain essential constituents of the diet, in agreement with the generally accepted etiology of true beri-beri, it should have made its first appearance before October among those who landed on the Peninsula in April, and it would be difficult to explain why it should have developed equally frequently among those of my cases who had been on the Peninsula for 6 to 16 weeks as among those who had been there for 20 to 24 weeks; I saw no cases among men who had been on the Peninsula for over six months. The supply of fresh food was more regular at Helles than at Anzac, and it was thought at first that the disease was least common at Helles, but this was not the case, as 11 out of the 17 patients (65 per cent.) came from Helles. Moreover many of the patients had had a fair supply of fresh bread and meat and occasionally even of eggs, articles of diet which would have prevented the development of beri-beri. An army baker developed the disease in spite of having had a very much more satisfactory diet than that of the majority of the troops. It is also noteworthy that the symptoms of neuritis generally did not begin until the patient had returned to a full diet for some days.

Lieut. C. R. N. Pattison has told me of a somewhat similar epidemic of peripheral neuritis following jaundice, which attacked about 80 out of the 300 lepers in the colony at Mokagai, Fiji, of

which he was acting superintendent, between June and August, 1914. The jaundice was followed in almost every case by pains in the calves and thighs, and difficulty or inability to walk with diminished or lost knee-jerks; the heart was greatly dilated, but there was no œdema, and three patients died of heart failure. He did not observe similar symptoms among any of the lepers who had not had jaundice.

It seems highly probable that the beri-beri, which occurred among British troops, was due at any rate in Gallipoli to a similar duodenal infection to that observed by Hamilton Wright, though deficiency in the anti-beri-beri vitamine was probably a contributory factor. The organism which infected the duodenum produced greater local swelling than in his cases, and it consequently caused jaundice in most cases with comparatively little damage to the nervous system, but the toxæmia occasionally caused symptoms indistinguishable from those of beri-beri. In most cases the interval between the onset of the duodenitis and the onset of the neuritic and circulatory symptoms was longer than in ordinary beri-beri.

In two fatal cases from Gallipoli the post-mortem appearances described by Willcox were very similar to those found by Hamilton Wright. The mucous membrane of the stomach was very red, especially in the pyloric half, where the colour was deep crimson. The duodenum showed intense crimson congestion of the mucosa, most marked in the upper part. The rest of the small intestine and the colon were also congested.

Symptoms.—The onset was generally rapid. In well-marked cases peripheral neuritis, very similar in character to that caused by alcohol, was associated with general œdema and dilatation of the heart, with a rapid pulse and dyspnœa on the least exertion. The earliest symptoms were generally weakness of the legs and shortness of breath, but these were occasionally preceded by œdema of the ankles.

The muscles of the legs and to a less extent of the arms were weak; the extensors were more affected than the flexors, so that foot and wrist drop were present and a "steppage gait" like that of alcoholic neuritis was often present. In severe cases walking was impossible. The muscles showed considerable atrophy and

were very tender on pressure, but the arms were less affected in this way than the legs. The deep reflexes were lost; in some of the earlier cases seen by Lieut.-Col. de Crespigny the knee-jerks disappeared before the ankle-jerks, but this was never observed among my cases, both jerks being absent in eight, both present in five, and the knee-jerks were present but the ankle-jerks lost in four.

Paræsthesia was common, the patient complaining of tingling or a sensation of pins and needles in his toes and fingers. More or less cutaneous anæsthesia and analgesia were always present and extended a varying distance up the legs; the arms were less frequently affected.

Œdema was most marked in the legs, but was also present over the lower part of the back, in the scrotum, over the abdomen and sternum and in the face in the severer cases. As a rule it disappeared much more rapidly than the other symptoms. In a few cases the beri-beri was of the "dry" form, with no œdema at all or only a little affecting the ankles.

The heart was dilated, and occasionally a systolic murmur was heard at the apex, but this disappeared as the condition improved. The pulse was rapid and sometimes irregular; the least exertion greatly increased its frequency and at the same time caused marked dyspnœa.

Pyrexia was generally absent, but in some cases slight fever continued for a few weeks.

Anorexia, slight epigastric discomfort, and in severe cases vomiting were often present, especially at the onset.

Case of Beri-beri following Jaundice.—Driver W. landed at Helles on May 6, 1915. He remained well in every way, complaining of no weakness, shortness of breath or other symptoms until October 16, when he suddenly lost his appetite. He felt sick when he saw food and he vomited when he tried to eat. On October 19 he became jaundiced, and on the 21st he was sent to the Lowlands Convalescent Depôt at Lemnos. He had had fresh bread daily and fresh meat five days a week whilst on the Peninsula until his illness began. After that he only had bread and condensed milk both at Gallipoli and at the Convalescent Depôt, with the addition of fresh meat on three occasions, and about two eggs a week. On November 15, when the digestive

symptoms were much better, the jaundice had almost disappeared, and he had been up for a few days, his legs felt stiff and weak and he became breathless and suffered from violent palpitation on the slightest exertion. On November 25 he was transferred to the 3rd Australian General Hospital. His legs, back, and face were œdematous, but there was no albuminuria; his pulse was 102, his heart was dilated and its action was thumping. The legs, and to a less extent the arms, were very weak; foot and wrist drop were well marked. The knee and ankle-jerks could not be obtained. The calves were very tender. There was some loss of tactile and painful sensation on the feet and round the mouth. He became progressively weaker for about ten days, but began to improve on December 10, soon after I first saw him with Lieut.-Col. Stawell. Between December 5 and 22 his temperature varied between 99° and 100°.

Arrangements were made for the patient to be transferred on his arrival in England to the care of Major Judson Bury, who reported on January 13, 1916, that the patient was much better, the cardiac symptoms having disappeared, and in May he was able to leave hospital almost completely cured.

Case of Beri-beri without preceding Jaundice.—Driver B. landed at Helles on May 9. He had dysentery for a fortnight in August; this was followed by diarrhœa, which continued on and off until he was sent to No. 2 Australian Stationary Hospital on November 5. He had never been jaundiced; he had had no appetite and had complained a good deal of abdominal pain with the diarrhœa, especially in the middle of October. He had had sixteen injections of emetine in August and September. On November 3 his legs began to get weak and he felt short of breath; on November 8 his legs became swollen. His heart was very dilated and his pulse rapid. His arms and legs became progressively weaker, till he had well-marked wrist and foot drop; his knee and ankle jerks were lost. His calves were very tender and there was definite anæsthesia of his feet and legs. His temperature rose almost every night whilst he was in hospital to between 99° and 100°. Slight improvement began to occur in the middle of December. Major Judson Bury reported on January 13, 1916, that there was not much change; the patient had slight movement at the knees and hips, none of the feet; he could feebly

extend his wrists. Anæsthesia was present up to the knees and there was well-marked hyperæsthesia of the muscles of the arms and legs, but no cutaneous hyperæsthesia. There was still some diarrhœa and abdominal pain. Soon afterwards he began to improve and left the hospital in May.

On August 22, 1916, the patient wrote to me that his legs are still weak, but he can now dorsi-flex his feet. His knee-jerks are absent, but there is now no anæsthesia. The arms, though thin, are quite strong. The ankles swell in the evening, but there is no palpitation or other cardiac symptom. He was fit enough to return to work in February, 1917.

The first of the above cases was selected as typical of the "beri-beri" following jaundice and the second as an example of the exceptional cases with no jaundice. It was afterwards discovered that the two men, who were in different hospitals in Lemnos, belonged to the same Field Ambulance at Gallipoli. The neuritis developed about the same time in both, and when one had jaundice, the other had noticed an aggravation in his abdominal pains, though no jaundice was present. It seems not unlikely that this aggravation represented an attack of duodenitis, the local results of which were insufficient to cause jaundice.

Prognosis.—Out of a total of approximately thirty well-marked cases from the Dardanelles three died from heart failure. In the others improvement began to take place after two or three weeks, but in the severer cases recovery was very slow.

Prophylaxis.—The occurrence of beri-beri in British soldiers shows the importance of providing special vitamine-containing food, which can be added to the ordinary ration, when troops are engaged in an expedition in which there may be difficulty in providing regular supplies of fresh food. This is particularly important if an epidemic of jaundice should break out, and all jaundiced soldiers should under these conditions be given a special supply of anti-beri-beri vitamine, both whilst they are actually ill and during convalescence. Haricot beans, pea soup made from pea powder, and porridge are valuable foods and are generally obtainable, but dried yeast or yeast cakes, which contain a specially large proportion of the necessary vitamine, should be carried with the medical equipment, and should be widely used if

an outbreak of beri-beri occurs and fresh food is unobtainable. Fresh vegetables and fruit should also be provided whenever possible in order to supply the vitamine necessary to prevent scurvy. The following list of articles of diet which contain the anti-beri-beri vitamine, was prepared by Lieut.-Col. W. H. Willcox and Major C. J. Martin; the articles are arranged in order, beginning with the one which contains the largest quantity: yeast, eggs (raw or lightly cooked), brain, liver, sweetbread, kidneys, heart muscle, peas, haricot beans, lentils, porridge, brown bread, fresh milk, fish and meat, ordinary bread and biscuits.

Treatment.—A generous mixed diet should be given with as large a proportion as possible of the articles already mentioned as containing the essential vitamine. In severe cases the indigestion and anorexia may make it impossible for a few days to take such a diet. Two ounces of "export yeast" or six "royal yeast cakes" should be given daily in such cases; the yeast is mixed with a little boiling milk into a cream, and then more warm milk and sugar are added (Willcox). If neither of these preparations is available, half a pint of yeast brew should be obtained from the army bakery and drunk after sweetening with sugar. Pea soup and three or four raw eggs beaten in milk should also be given.

The patient should rest in bed until his heart is no longer dilated and the pulse rate is normal. It is doubtful whether any drugs assist the heart, but digitalis may be tried in full doses. Exercises should be begun at an early date for the arms and subsequently for the legs, as apart from their value in hastening recovery from the paralysis they are very useful in restoring tone to the cardiac muscle.

The treatment of the paralysis does not differ from that caused by other forms of neuritis. It is most important to prevent the paralysed muscles becoming over-stretched whilst the patient is still in bed; the hands should be kept extended by splints if wrist-drop is present, and the feet dorsi-flexed if there is foot-drop. Massage and passive movements should be begun as soon as the muscular tenderness has sufficiently subsided.

References

Hamilton Wright: *British Medical Journal*, 1905, II. 1095.
W. H. Willcox: *Lancet*, 1916, I. 553.

CHAPTER VIII

SOLDIER'S HEART

Soldiers not infrequently suffer from symptoms due to functional circulatory disturbances during their period of training and still more often whilst on active service. In neither case do the symptoms differ from those which may occur among civilians, but their relative frequency has led to the adoption of the term "soldier's heart" to describe the various functional cardiac disorders specially common among soldiers. The effect of active service on the heart was first investigated during the American Civil War, and the present war has given an opportunity for a renewed study of the subject. Some confusion has arisen from the fact that different writers have described different conditions under the same designation, and that there is a tendency for each observer to regard the particular form of cardiac disorder in which he is specially interested as the only one of sufficient importance to be called the "soldier's heart." Although much new light has been thrown on the subject, the different varieties of soldier's heart cannot yet be defined with sufficient accuracy to make it possible to give a separate description of their etiology, symptoms and treatment.

Etiology.—Soldier's heart is most commonly due to the effect of over-exertion, which may be associated with prolonged mental strain and insufficient sleep, on a heart and nervous system weakened by the action of some form of toxæmia. Although physical strain, mental strain, and toxæmia generally act together, I have seen many cases in which one or other of these factors could be definitely excluded.

(*a*) *Intoxication.*—(i) The toxæmia is most frequently bacterial in origin, the symptoms dating from some preceding infective

disease. This is sometimes a definitely recognised infection: dilatation of the heart and tachycardia may develop in the course of paratyphoid fever; epidemic jaundice is often accompanied by signs of cardiac poisoning and was a common cause of soldier's heart at Gallipoli; trench fever appeared to have little effect on the heart until the hot weather began in Salonica, when it was very frequently followed by cardiac symptoms associated with a moderate degree of dilatation. Bacillary dysentery may be followed by cardiac weakness, but less frequently than the non-specific intestinal disorders associated with diarrhœa and little or no rise of temperature, in which the nature of the infection is obscure. A history of tonsillitis and occasionally of measles or scarlet fever may be obtained. It is, however, often impossible to ascertain the nature of the infection, which may have been described as "influenza," though true influenza has been rare among troops on active service, or simply as "pyrexia of unknown origin." In some cases the infection is not sufficiently severe to cause the soldier to report sick, and keen men often continue at duty in spite of having a febrile illness, which ought to be treated by complete rest.

(ii) I am convinced that the toxæmia which results from excessive smoking is often a very important factor. Many soldiers, especially those under twenty years old, smoke much more than they did before they joined the army, and the custom of giving cigarettes to convalescent soldiers, whilst they are in hospital recovering from various infections, is responsible for much subsequent trouble, as the heart and nervous system, already poisoned by the toxins produced by the infection, are particularly liable to be further damaged by the toxins inhaled whilst smoking.

(iii) In a small proportion of cases the intoxication is due to the excessive activity of the ductless glands, which results from the nerve strain of active service. The fact that over-activity of the thyroid gland can be more easily demonstrated than that of other glands has led to these cases being regarded as examples of simple hyperthyroidism, but there is little doubt that the condition is really a highly complex one, in which there is a disturbance in the activity of all the ductless glands, especially the suprarenal and thyroid, so that some such name as the "hormonic neurosis" would be more accurate. Cannon and Crile have shown experimentally that fear, exhaustion from fighting, and acute infections increase

the secretion of adrenalin and in time lead to exhaustion of the suprarenal glands.

(*b*) *Over-exertion.*—Over-exertion is a relative term. A well-trained man can do work which would be impossible in the early stages of his training, and which would again be impossible if his heart and nervous system became damaged by the toxins produced by an infection, excessive smoking, or excessive activity of the thyroid and other ductless glands. The irritable heart of the recruit, which was at one time ascribed to restriction of the thoracic movement by the accoutrements and at a later date to a badly devised form of drill, but continued to occur with undiminished frequency after the accoutrements and drill had been altered, is to a large extent a result of attempting to train too rapidly. It is as common in the big guardsman who has outgrown his strength as in the under-developed and under-fed recruit from the slums. Skilful and graduated training would prevent the development of an irritable heart in peace time as in war. A history is occasionally obtained from officers that they suffered from somewhat similar symptoms when fifteen or sixteen years old after too strenuous indulgence in long-distance or cross-country running, and men frequently admit that they have often in the past had similar but generally less severe symptoms on taking unwonted exercise.

The trained soldier rarely develops cardiac symptoms as a result of over-exertion whilst on active service, unless the toxic factor is also present. But with the nervous system and heart enfeebled by the toxæmia, the physical exertion and mental strain of life in the trenches, which hitherto produced no ill-effects, are sufficient to cause nervous exhaustion and cardiac weakness.

(*c*) *The nervous factor.*—The nerve strain of active service affects the circulation, probably through the intermediation of the ductless glands, the activity of which may be so profoundly modified by prolonged strain to the nervous system that the condition already referred to as the hormonic neurosis with its circulatory and other symptoms may result. But the nervous system may affect the circulation more directly. One of the worst cases of "soldier's heart" I have seen developed in a previously healthy soldier immediately after he regained consciousness after being blown up by a shell; the symptoms were almost as severe as ever when I first saw him twenty-two months after

the onset, although he had been invalided out of the army three months before. His condition rapidly improved with suggestion and graduated exercises. I saw a soldier in Salonica who was suffering from severe paroxysmal attacks of tachycardia, especially at night. On cross-examination he admitted that they were invariably caused by a dream or the sudden recollection when awake of the horror he had experienced a year and a half before, when he had found the dead body of a man in the bedroom of his billet.

The soldier's heart is often only one manifestation of the nervous exhaustion or neurasthenia, which results from the combined effects of physical fatigue, mental strain and toxæmia. The neurasthenia gives rise to an excessive irritability of the central nervous system, which causes slight circulatory disturbances to produce precordial discomfort, pain and palpitation, although the actual condition of the heart is such that no subjective symptoms would occur in a man with a normal nervous system.

Symptoms.—As already mentioned, the onset of symptoms often dates from some febrile attack, the patient having returned to duty before he felt completely fit. In many cases actual cardiac symptoms are preceded, often for some weeks, by neurasthenic symptoms, which are due to the effect of toxins on the central nervous system, the resistance of which is frequently already weakened by prolonged physical and mental strain with insufficient rest. A moderate amount of exercise, which would formerly have produced no ill-effect, now gives rise to shortness of breath and exhaustion, which may be accompanied by giddiness and faintness. The patient feels unfit even when at rest, and he is often irritable and depressed about his health. The vasomotor centre is particularly affected, so that the hands and feet are always cold: at one time the fingers are shrivelled, white or blue, and numb; at another they are swollen, red, and tingling.

More violent exertion causes dyspnœa and palpitation, which may last for some hours; after a time the patient gets out of breath after gentle exercise, and palpitation may be troublesome even when he is at rest, especially on first getting into bed at night. Occasionally slight swelling of the ankles is present, but

œdema is never a prominent symptom. Discomfort, which may amount to severe pain, is often felt in the neighbourhood of the heart, not only on exertion but also when at rest. This is likely to make the patient worry about himself, as he begins to think that his heart must be seriously diseased. In some cases, especially when the condition is in part due to excessive smoking, there is persistent tachycardia. More frequently the pulse is normal or only slightly increased in rate whilst resting, but the least exertion or excitement unduly accelerates it, and several minutes elapse before it returns to normal; sitting up in bed or standing may change a pulse of 70 to one of 100. The blood-pressure is normal or low except in the hormonic neurosis, which Colonel A. E. Garrod and Major G. Graham have found to be constantly accompanied by hypertension.

In most cases there is some dilatation of both sides of the heart, especially the right; occasionally it is extreme. This is due to atony of the poisoned heart muscle. The simultaneous dilatation of the auriculo-ventricular ring may result in mitral incompetence, shown by the presence of an apical systolic murmur, and the dilatation of the conus arteriosus gives rise to a systolic murmur in the pulmonary area, which is sometimes very loud and widely propagated. The presence of these murmurs often leads to the diagnosis of valvular disease of the heart, which is not only incorrect, but has also a very unfortunate effect on the mind of the patient. The murmurs are always systolic, and are generally much louder in the recumbent than in the erect position, in which the heart becomes elongated and the dilated chambers narrowed; in some cases they disappear completely on standing.

In cases of so-called "hyperthyroidism" or the "hormonic neurosis," the pulse is rapid, especially on the slightest exertion, and the heart may be slightly enlarged, but there are generally no murmurs. Vasomotor instability is well marked; if the handle of a spoon be drawn across the skin a white band with red borders appears and may remain for some minutes. Excessive sweating is common, and the patient generally loses weight. The hands and occasionally the eyelids are tremulous, and the patient is often highly nervous and excitable; the eyes may be slightly prominent and von Graefe's sign may be obtained. The thyroid gland is often, but not invariably, enlarged, but never to a great

extent. The blood-pressure is always raised, being often 150 or 160 mm. of mercury in a man of twenty-five. This points to other ductless glands, such as the pituitary and suprarenal, being involved in addition to the thyroid, as the blood pressure is not raised in uncomplicated Graves' disease.

Diagnosis.—The functional disorders grouped together as soldier's heart must be differentiated from the three organic conditions which may cause cardiac symptoms in young men—infective endocarditis, chronic valvular disease and angina pectoris; the first and last of these are, however, very rare in soldiers In the functional disorders the temperature is generally normal, although there is often a history of an earlier pyrexial attack, and in some cases the infection is so chronic that a slight degree of pyrexia continues for many weeks. In infective endocarditis, on the other hand, the temperature is either constantly or intermittently raised. Whereas any murmurs which may be present in the soldier's heart are always systolic, and tend to become less marked as time goes on and the patient's condition improves, diastolic as well as systolic murmurs are often present in infective endocarditis, and new ones may develop owing to organic changes occurring in the valves; they tend to vary in character from week to week and to become more and more marked. Symptoms of infarction in various organs may occur, and the general condition of the patient is more serious. In chronic valvular disease there is almost always a history of true rheumatic fever or occasionally of scarlet fever. Murmurs may be diastolic as well as systolic; they do not become fainter but may actually become louder as the patient's condition improves, more or less hypertrophy is present, which varies with the exact lesion, and the murmurs are as loud in the erect as in the recumbent position: the systolic apical murmur is traceable further into the axilla and often to the back, and pulmonary systolic murmurs are rare except in congenital heart disease. In the poisoned heart of soldiers a feeling of exhaustion often precedes the cardiac symptoms, whereas in chronic valvular disease dyspnœa is generally the first symptom and œdema is much more common. Angina pectoris is rare in young men, in whom it is almost always due to syphilis; pain, which may be the only symptom, at first only occurs on exertion.

Prognosis.—Men of feeble physique, who even in civil life have been unable to take much exercise without becoming dyspnœic and experiencing pain in the cardiac region, and have never been really fit from the time they joined the Army, are unlikely to improve sufficiently to make useful soldiers. With this exception the prognosis is good, even in apparently severe cases, and very few men should be invalided out of the Army for soldier's heart. Complete recovery requires treatment for a period varying between a fortnight in the mildest cases and three to nine months in severe cases.

Prophylaxis.—Feebly developed recruits, who have hitherto followed a sedentary occupation and have taken comparatively little exercise, should not be trained too rapidly. Skilful handling of recruits results in many men, who would otherwise soon complain of cardiac symptoms, becoming strong and capable of considerable endurance. Soldiers should not be allowed to smoke excessively, and when suffering from any febrile disorder or from the slightest cardiac disturbance they should not be allowed to smoke at all until they have completely recovered—and then only in strict moderation. Care should be taken not to allow convalescents from infective disorders to get up too soon. The heart should be frequently examined, and if any dilatation is observed the patient should be kept in bed until it has almost, if not completely, disappeared. The tonsils should be enucleated if recurrent attacks of acute tonsillitis occur, as this often leads to poisoning of the heart muscle.

Treatment.—The first essential in the treatment of soldier's heart is to encourage the patient and to convince him that his heart is not really diseased and that he will completely recover his health. In the numerous cases which have already been erroneously diagnosed as V.D.H.—valvular disease of the heart—this may be exceedingly difficult. It is therefore of the utmost importance that this diagnosis should only be made when there is no possibility of mistake about it. Even the commonly accepted official diagnosis of soldier's heart—D.A.H. or "disordered action of the heart"—is best avoided, as when the word "heart" has once been employed it becomes extremely difficult to eradicate the idea of heart disease from the soldier's mind, and it is

comparatively rare for him to become an efficient fighting man again, although the actual cardiac condition is generally quite curable. For this reason I believe that some such diagnosis as "debility" is preferable; it is a perfectly correct one, as there is no doubt that the functional capacity of other parts of the body as well as the heart is impaired. In many cases the cardiac condition is simply part of general neurasthenia, and a diagnosis of neurasthenia would be quite satisfactory, were it not for the unfortunate tendency of some medical officers to regard neurasthenia as synonymous with malingering. The "hormonic neurosis" is probably most conveniently diagnosed under the more simple but less accurate name of "hyperthyroidism."

In the acute stage rest in bed is necessary, but this should never be prolonged; even if the heart is still much dilated and the pulse rapid the patient should be allowed to get up after a short initial period of rest, so long as the temperature is normal. He should be given graduated exercises, which should always be just insufficient to cause exhaustion, dyspnœa or pain. Walking exercises first on the level and then on hills of increasing steepness are of great value. Major C. H. Benham organised graduated exercises of this sort for soldiers with functional cardiac disorders at the 29th General Hospital in Salonica, and the results were very satisfactory. Whenever possible the exercise should be of a congenial nature and taken in the open air with a cheerful companion. Officers should be encouraged to golf, shoot or ride, whichever they prefer, and they should always rest for a time if they feel tired, out of breath or otherwise unwell. In convalescent hospitals non-commissioned officers and men with soldier's heart should not only be encouraged to play games, but they should be given a regular occupation, such as gardening, farm-work, or carpentering, for a prescribed length of time twice a day, and at a still later stage regular drill of gradually increasing duration is most valuable. A man should not be sent back to his unit until he is able to do an average day's work.

Cardiac stimulants, such as digitalis, do no good and may do harm. The only drug which is often useful is sodium or potassium bromide, which should be given in small doses, such as gr. v, two or three times a day for several weeks, in the numerous cases in which the nervous system is irritable. A small dose of some

hypnotic, such as medinal, with gr. x of acetyl-salicylic acid, will generally procure sleep if insomnia is present. If this is not effective, and especially if the patient is much worried by palpitation at night, treatment by suggestion is almost always successful.

The hormonic neurosis is best treated by rest and small doses of bromide. X-ray applications to the thyroid gland have been used with success in some cases, but it is impossible to regulate the destructive action of the rays with sufficient accuracy for perfect safety, and the other ductless glands are not dealt with. The nervous condition of the patient is often greatly benefited by suggestion; I was able to observe the good effects of hypnotism on cases of this sort treated by Captain J. B. Tombleson in Malta, and I have since had similar experience myself.

References

H. Harthorne: *American Journal of Medical Science*, XLVIII. 89, 1864, and da Costa, *American Journal of Medical Science*, LXI. 17, 1871. (Soldier's heart in the American Civil War.)

Discussion on the soldier's heart (Sir James Mackenzie, R. M. Wilson, F. J. Poynton, R. W. Mitchell, A. Morrison, Florence A. Stoney, and others), *Proc. Roy. Soc. Med.*, Therap. Section, IX. 26, 1916; also *Brit. Med. Journ.*, 1916, I. 117; *Lancet*, 1916, I. 777.

G. W. Crile: *The Origin and Nature of the Emotions*, 1915.

J. Parkinson: *Lancet*, 1916, II. 134.

J. C. Meakins and others: *Brit. Med. Journ.*, 1916, II. 418.

CHAPTER IX

WAR NEPHRITIS

Acute nephritis is a comparatively rare disease in civil life, an average of 13 male cases, only 5 of which were in men of military age, being admitted each year into St. Bartholomew's Hospital out of a total of 7000 patients. It does not appear to have been common in any previous campaign except the American Civil War, in which over 14,000 soldiers of the northern armies were invalided for nephritis. Up to June, 1915, there had been 1062 cases in the British Army in France and Flanders, a very large proportion of which had occurred since March. Very few cases occurred among French troops until the autumn, by which time the infection appears to have spread from the neighbouring British lines. There was a similar, but less severe outbreak in the Gallipoli and Salonica Armies, and the disease, which has also been very prevalent among French, German and Austrian soldiers since the spring of 1915, is very rare among officers of all nationalities.

Definition.—War nephritis is a form of acute nephritis, which has occurred among soldiers on active service during the present war ; it differs in its great frequency, its comparatively favourable course and the constancy of certain clinical characteristics from the acute nephritis observed among young adults in civil life. The condition has been called "trench nephritis," but the name is unsuitable, as there have been many cases in men of the Army Service Corps, ammunition columns, headquarters troops and R.A.M.C., who have never been in the trenches.

Etiology.—The majority of cases occur among soldiers who were previously healthy. There is no doubt that a former attack of nephritis renders an individual more liable to develop

the disease, which also tends to be unusually severe. In a small number of cases a definite history of a previous attack of nephritis has been obtained, and in a few others the clinical symptoms or the autopsy points to the existence of chronic nephritis.

Exposure to cold or wet aggravates war nephritis when it has once developed, and may produce an acute attack in men whose kidneys are already damaged, but it is certainly not an important factor in the large majority of cases. The disease was rare in France during the first winter of the war in spite of the frequency of exposure to cold and wet; it only became common in March and April, during which months more cases occurred than in the preceding seven months, and the high incidence continued through the summer. The majority of cases of nephritis which occurred before March, 1915, were due to acute exacerbations in middle-aged men with renal disease, and were not genuine examples of war nephritis at all. Sudden changes in temperature also appear to be of small importance, as in the present war such changes have never been as extreme as in the South African and Russo-Japanese wars, in both of which nephritis was very rare. If cold were an important factor, Indian troops would probably suffer greatly from the disease, but only three cases were reported among them; this, however, is perhaps due in part to the protection against chills afforded by the loin cloth they constantly wear.

The nephritis might conceivably be due to metallic poisoning caused by the consumption of tinned rations, especially those which are cooked in the tins, although MacKenzie Wallis could find no trace of metallic poison in the urine; Powell White, using a different method, found lead in the urine of each of four cases of war nephritis which he examined, and tin in one of them. The latter was the most recent case and more lead was present than in the others; a month later there was less lead and no tin. Powell White found that salt solution boiled in a fruit or meat tin extracts traces of lead and tin, the amount being greatly increased if the slightest trace of acid is present. Further investigations are required in order to show how much importance can be ascribed to these observations. War nephritis is certainly not due to the chlorination of water, as it has been just as common

among men who have never had any as among those who have constantly drunk it. In the Austrian army soldiers have purposely taken cantharides and chromic acid in order to produce nephritis, on account of which they hoped to be discharged.

Acute tonsillitis and other infections may be followed by nephritis among soldiers as among civilians, but this only accounts for a very small proportion of the cases. Cultures from the throat in ten soldiers with nephritis did not differ from cultures from ten wounded soldiers, and in both cases the throat was less septic than in ten civilian patients who were in the same hospital for various surgical conditions (R. G. Cantie). Eighteen out of fifty-six cases gave a positive Wassermann reaction (Mackenzie Wallis); the proportion is very much greater than would be found in the Army generally, so that syphilis, which under normal conditions gives rise to albuminuria in 4 per cent. of cases during the secondary stage, appears to predispose to war nephritis.

Diarrhœa frequently preceded the onset of nephritis in the troops at Gallipoli, but this was probably accidental and due to its extreme frequency, as no such history was obtained in the majority of cases occurring elsewhere. The urine is generally sterile; in a few cases the *B. coli* has been isolated, but this was probably due to coincident intestinal disturbances and is not likely to be of any pathological significance. The absence of urobilin and of any increase in ethereal sulphates in the urine is also against an intestinal infection. Bronchitis often precedes or accompanies the nephritis, but it was common among Indian troops in spite of their freedom from nephritis.

As the majority of cases occurring both in France and in the Eastern Mediterranean Forces resemble each other closely, whilst differing in many respects from the ordinary acute nephritis seen in civil life, it seems probable that the disease is due to a specific infection, which is perhaps identical with that which caused the epidemic in the American Civil War.

Morbid Anatomy.—In the very small number of fatal cases in which there was no evidence of old kidney disease, the glomeruli, tubules and interstitial tissue were all affected, the condition being indistinguishable from the acute nephritis following scarlet fever; there was no pyelitis.

Symptoms.—The first symptoms are generally headache, œdema of the face and ankles, and breathlessness. The headache may be sufficiently severe to prevent sleep; it is sometimes accompanied by vertigo. Œdema, which was uncommon in the cases which occurred before March, 1915, is almost always present, but it is rarely universal and never sufficient to require puncture; slight ascites sometimes occurs, but hydrothorax is rare; pulmonary œdema is common. The breathlessness is a very constant and characteristic early symptom; it is at first only present on exertion, but it generally continues after the patient is taken into hospital, when it tends to be most marked at night. There is often some pain in the lower part of the back and the limbs; I have frequently noticed tenderness in the renal region on both sides, and in some cases the kidneys were palpable and appeared to be enlarged as well as tender. The temperature is generally raised to 100° or 101° at the onset, sweating is much more frequent than in other forms of nephritis, and herpes labialis may occur. Bronchitis is often present and may cause a troublesome cough. The headache and perhaps the dyspnœa may be regarded as uræmic symptoms; nausea, vomiting, slight drowsiness and apathy are not uncommon. Epileptiform convulsions sometimes occur suddenly in apparently slight cases, but the blood-pressure is then always high. An ammoniacal odour is often noticeable, and in one case of Bradford's the patient complained of a taste of ammonia. Amaurosis and transitory acute mania have been observed. Cutaneous eruptions are rare, and cramps and hissing dyspnœa do not occur.

The blood-pressure is always found to be raised if taken in the evening. Those observers who have found the rise less constant have probably taken their records in the morning, as Captain R. G. Abercrombie has shown that there is a considerable diurnal variation in this form of nephritis, the pressure at 6 p.m. being often 20 to 60 mm. Hg. higher than at 10 a.m. The increased severity of the headache and dyspnœa at night are probably connected with the rise in blood-pressure. The increased pressure often only lasts from five to ten days, but in some cases it may continue for a few weeks. The morning systolic pressure is generally between 135 and 180 mm. Hg.; it is highest when uræmic symptoms, especially convulsions, are prominent, and it may then be even

200 mm. Hg. The heart is not dilated and hypertrophy rarely develops. Albuminuric retinitis only occurs in cases with persistent albuminuria and a high blood-pressure, when the disease appears to be becoming chronic. Pleurisy and pneumonia are rare, and pericarditis has not been recorded.

The urine is at first scanty, especially when there is much œdema, and uræmic symptoms may be accompanied by suppression for twelve or twenty-four hours; the quantity of albumin varies greatly in different cases and from day to day in the same case, but it is often considerable at first. Casts are generally found in large numbers; the majority are hyaline and granular, and there are sometimes a few epithelial casts, but fatty and blood casts are uncommon. A varying and occasionally considerable amount of blood is present; fever is generally well marked and œdema slight or absent in cases in which the urine is at first smoky or red. There is often a good deal of pus; this is probably of renal origin, as infiltration of the interstitial tissue of the kidneys with similar polymorphonuclear leucocytes is always found in fatal cases, and definite evidence of pyelitis is rarely present. A similar condition of the urine and the kidneys is very unusual in acute nephritis with the exception of that due to scarlet fever.

Improvement occurs rapidly. The temperature generally falls in a few days, but may remain slightly raised for some weeks in the more prolonged cases. Sometimes the œdema only lasts two or three days; it generally disappears within ten days and is rarely present longer than a fortnight. Uræmic symptoms disappear within the same period; the blood-pressure falls, the oliguria is replaced by polyuria, and the abnormal constituents of the urine diminish in quantity, disappearing completely in three or four weeks in most cases, although a little albumin, a few casts, and occasionally a good deal of blood may remain for a considerable period after all other symptoms have disappeared. Sometimes the albuminuria is intermittent before it finally disappears. In other cases blood may reappear in the urine and the albumin increase in quantity; other symptoms may return, but œdema is rare.

Diagnosis.—Before labelling a case of nephritis in a soldier as "war nephritis," it is necessary to exclude the ordinary causes, such as scarlet fever, acute tonsillitis, and in rare cases typhoid

and paratyphoid fever, malaria, and dysentery. Owing to the frequency of syphilis among soldiers, it is probable that some cases diagnosed as war nephritis are really cases of syphilitic nephritis; the recognition of such cases is extremely important, as they promptly respond to treatment with mercury and iodides, whereas mercury generally aggravates the symptoms and increases the albuminuria in nephritis due to other causes.

Young soldiers are occasionally sent from their units to hospitals as cases of nephritis, but on examination of the urine no albumin is found. These are probably examples of "functional albuminuria" and not nephritis at all; a man may have fallen out during a march on account of faintness, and examination of the urine has shown the presence of albumin. This is simply due to excessive exertion, and is comparable to the temporary albuminuria which frequently occurs after athletic contests. McLeod and Ameville found that the incidence of albuminuria in apparently healthy British and French troops was between 1·6 and 4·7 per cent. under various conditions in France, but was as high as 10·1 per cent. among recent recruits during strenuous training in England. Such cases can be sent back to their units after resting for two or three days, as this form of albuminuria does not in any way predispose to nephritis.

The characteristic features of war nephritis are the transitory and comparatively slight œdema, the frequency of dyspnœa, the rarity of inflammatory complications and of the pale waxy swollen face characteristic of ordinary acute nephritis, and the low mortality in spite of the liability to sudden attacks of severe uræmic convulsions. It is important to distinguish war nephritis from acute exacerbations of ordinary chronic renal disease; the latter occurs as a rule in older men, the heart is hypertrophied, the blood vessels thickened, retinal changes are present, and œdema may be slight or absent.

Prognosis.—Death is extremely rare, not more than three or four patients dying per 1000; it is almost always due to uræmia, and in most cases chronic nephritis or a congenital abnormality of the kidneys has been found at the autopsy; in one case it was due to coincident broncho-pneumonia. Most patients get apparently well within four weeks, but complete recovery may

occur even after six months. The ultimate fate of those in whom slight albuminuria persists for many months is still doubtful, but it seems probable that the majority of cases which last over twelve weeks become chronic.

Treatment.—Rest in bed, warmth, and a diet consisting of milk, milk puddings and bread and butter are all that is required in most cases. A larger quantity of nitrogenous food should only be allowed when the headache and dyspnœa have disappeared; an ordinary diet can then be given whatever the condition of the urine.

Whenever possible a Wassermann reaction should be done, and mercury and iodide given if it is positive; when this is impossible, the same treatment should be tried if there is the least possibility of a syphilitic origin, but it should not be continued for more than five days if no definite improvement results. Any local infection, such as inflamed tonsils, sinus disease or diarrhœa should of course be treated.

Severe headache can be relieved instantaneously by lumbar puncture. Uræmic convulsions can be controlled by chloroform, and their return prevented by morphia, lumbar puncture, and by bleeding; in the exceptional cases in which the blood-pressure is not raised, saline solution should at the same time be injected intravenously. I have never seen any benefit follow the use of hot-air baths, and in three cases mentioned by Abercrombie fits appear to have been brought on by hot packs.

Owing to the danger of relapse on exposure to the hardships inseparable from active service, a man who has once suffered from acute nephritis should never return to more than light duty at home. Possibly, however, an exception could be made for very slight cases, which appear to recover completely within two or three weeks.

References

British Medical Journal, 1915, II. 109, and 1916, I. 251.
W. Langdon Brown: *Lancet*, 1916, I. 391.
Proc. Roy. Soc. Med., Med. and Therap. Sections, IX. Feb. 15th, 1916.
Sir J. R. Bradford: *Quarterly Journal of Medicine*, IX. 125, 1916.
B. W. Hogarth: *Journal of the R.A.M.C.*, xxvi. 372, 1916.
R. L. Mackenzie Wallis: *Journal of the R.A.M.C.*, xxvi. 259, 1916.
C. Powell White: *Lancet*, 1916, I. 996.
R. G. Abercrombie: *Brit. Med. Journ.*, 1915, II. 531, and 1916, I. 877; and *Journal of the R.A.M.C.*, xxvii. 131, 1916.
J. W. McLeod and P. Ameville: *Lancet*, 1916, II. 468.

CHAPTER X

GAS-POISONING

Poisonous gases are produced by all explosives, but owing to their rapid diffusion the majority are harmless unless they collect in closed spaces such as dug-outs or cellars. Under such conditions carbon-monoxide poisoning may occur (*vide* shell-shock). Shells which are specially constructed to set free asphyxiating and lachrymatory gases have been much used, but very little is known about their composition. The most serious form of gas-poisoning and the only one which will be considered in this chapter is that produced by heavy gases, which are set free in the enemy's trenches to drift with the wind. This brutal method of warfare was clearly premeditated, as shortly before the war a leading German pharmacologist and his pupils published papers which show that they were investigating the subject in great detail.

Asphyxiating gas was first used on April 22nd, 1915, in a German attack on Algerian and Zouave troops, who, being taken completely by surprise, broke before it. On the two following days attacks under cover of gas were made on Canadian and English soldiers in the neighbourhood of Ypres. Although no respirators were available and the losses were heavy, the troops held their ground. Another attack was made early in May; the men had now been provided with respirators, consisting of pads soaked in a solution of sodium bicarbonate, and these gave some protection. The third attack on May 24th and all subsequent ones have produced less serious results, as respirators of steadily increasing efficiency have been introduced.

The gas used has never been collected for analysis, but it is probably chlorine in most cases; indeed a deposit of chlorides has been found on the buttons of gassed soldiers. Chlorine was chosen on account of its exceptionally irritating character; it rapidly puts a man out of action when inhaled in a strength of only 1 in

10,000, whereas sulphur dioxide is only effective in a concentration four times as great. Chlorine, being much heavier than air, readily drifts and sinks into trenches, dug-outs, and cellars. It can be rapidly manufactured in enormous quantities and is easily compressed into cylinders, in which it can be conveyed to the front.

The first effect produced by the irritant action of the gas is a profuse exudation of a thin, light yellow, albuminous fluid by the bronchial mucous membrane, as well as a very active secretion by the lachrymal and salivary glands; these are the results of protective reflexes, the object of which is to dilute the irritant poison and render it innocuous. At the same time spasm of the bronchial muscles occurs in an attempt to obstruct the passage of the gas into the alveoli. In severe cases the bronchial secretion and spasm not only fail to protect the alveoli, but obstruct the entry of air into the lungs, to such an extent that the patient becomes asphyxiated and may die before the fluid is expectorated and the spasm relaxes. An autopsy at this stage shows slight congestion of the larynx and intense congestion and œdema of the trachea and larger bronchi, which are filled with frothy fluid. The lungs are intensely congested and œdematous, but the violent respiration caused by the asphyxia produces small patches of over-distended lung, seen on the surface as light grey areas in the least damaged parts, into which air can still pass. The distended alveoli may rupture into the interstitial tissue, and air may spread into the mediastinum and even to the neck.

In all but the mildest cases the asphyxial stage is followed by a stage in which acute inflammation with profuse exudation of lymph occurs as a result of the irritant action of the gas on the bronchial mucous membrane and the alveoli. If the patient dies in this stage the serous fluid in the bronchi is replaced by muco-pus, and more or less extensive broncho-pneumonia is found.

There is no conclusive evidence that the chlorine is absorbed by the blood and conveyed by it to other parts of the body. Nephritis has occasionally been found post-mortem, though there has very rarely been any clinical evidence of its presence; thus albumin and casts are rarely found, œdema never occurs, and only one case of uræmia has been recorded. According to Leonard Hill, the nephritis is not due, as suggested by Bradford and Elliott, to

the toxic action of the gas after absorption; he regards it as a result of asphyxiation and analogous to the condition which results from temporary occlusion of the renal artery. At a later stage secondary toxic effects may be caused by absorption of the products of the pathological changes in the lungs. If death occurs in the earlier stages, the right side of the heart is greatly dilated and the brain and all the abdominal organs show marked congestion due to asphyxia. The mucous membrane of the stomach is red and covered with thick yellowish mucus, submucous hæmorrhages are common, and superficial erosions may be present: these changes are partly due to the asphyxia and partly to the irritant action of chlorine, dissolved in swallowed saliva and nasal and bronchial secretion.

Symptoms.—The first effect of inhalation of chlorine is a burning pain in the throat and eyes, accompanied by a sensation of suffocation; pain, which may be severe, is felt in the chest, especially behind the sternum. Respiration becomes painful, rapid, and difficult; coughing occurs, and the irritation of the eyes results in profuse lachrymation. Retching is common and may be followed by vomiting, which gives temporary relief. The lips and mouth are parched and the tongue is covered with a thick dry fur. Severe headache rapidly follows with a feeling of great weakness in the legs; if the patient gives way to this and lies down, he is likely to inhale still more chlorine, as the heavy gas is most concentrated near the ground. In severe poisoning unconsciousness follows; nothing more is known about the cases which prove fatal on the field within the first few hours of the "gassing," except that the face assumes a pale greenish yellow colour. When a man lives long enough to be admitted into a clearing station, he is conscious, but restless; his face is violet red, and his ears and finger-nails blue; his expression strained and anxious as he gasps for breath; he tries to get relief by sitting up with his head thrown back, or he lies in an exhausted condition, sometimes on his side with his head over the edge of the stretcher in order to help the escape of fluid from the lungs. His skin is cold and his temperature subnormal; the pulse is full and rarely over 100. Respiration is jerky, shallow and rapid, the rate being often over 40 and sometimes even 80 a minute; all the

auxiliary muscles come into play, the chest being over-distended at the height of inspiration and, as in asthma, only slightly less distended in extreme expiration. Frequent and painful coughing occurs and some frothy sputum is brought up. The lungs are less resonant than normal, but not actually dull, and fine râles with occasional rhonchi and harsh but not bronchial breathing are heard, especially over the back and sides.

Headache is generally severe, and there is also considerable epigastric discomfort, due partly to the strain of coughing and partly to gastric irritation, as it is increased if an attempt is made to eat.

The intense dyspnœa of this asphyxial stage lasts about thirty-six hours, after which it gradually subsides, if death does not occur before. The patient, exhausted from his fight for breath, then falls asleep and wakes up feeling much relieved.

A few hours later acute bronchitis or broncho-pneumonia develops. In severe cases the quiescent interval is short and the bronchitis very severe. The sputum is now viscid, yellow or greenish, and muco-purulent with occasional streaks of blood. Respiration becomes more shallow and rapid, and the rate may finally be even 70 or 80 a minute. The pulse is small and very rapid; the temperature rises, and is often as high as 104°. The patient may now become delirious. Pleurisy may occur, and in some instances empyema and gangrene of the lung have followed.

After recovery from the bronchitis and pneumonia the patient remains weak and exhausted for a considerable time. He gets tired very rapidly and is unable to walk quickly or up hill without getting short of breath, even after the last signs of bronchitis have disappeared. He may continue to have attacks of dyspnœa and cyanosis for several weeks. The frightful experience he has passed through often affects his nervous system, and some of the attacks are doubtless aggravated by apprehension. Headache, vertigo and dyspepsia may continue for several weeks.

Prognosis.—Nothing is known as to the proportion of men who die from "gassing" on the field. Before efficient respirators were in use about 5 per cent. of those who reached the clearing stations died within forty-eight hours. Of those who reached the base hospitals between 1 and 2 per cent. died in the second or third week from broncho-pneumonia or other pulmonary complications.

The mildest cases are often fit for light duty after a short period of rest, but they should not be sent back until all adventitious sounds have disappeared from the lungs. A considerable time elapses before complete restoration of health occurs in the more severe cases, and it is still doubtful whether more or less permanent incapacity may not sometimes follow.

Prophylaxis.—The introduction of efficient respirators has almost abolished the danger of drift gas. Regular drill in the use of the respirators and inspection to see that they are in good condition are most important, as it takes time to get accustomed to breathing whilst wearing a respirator, and a damaged respirator may be worse than useless.

Treatment.—The patient should be kept warm with extra blankets, hot-water bottles and hot drinks, and his bed should be near an open window or out of doors. Owing to the irritated condition of the stomach a fluid diet should be given at first. Absolute rest is of the greatest importance.

In severe cases the chief object of treatment is to expel the fluid, which is drowning the patient, from the lungs. This can be done by artificial respiration, repeated whenever the dyspnœa becomes excessive. After squeezing the fluid out of the lungs, it may be necessary to blow air in from mouth to mouth in order to overcome the resistance of the froth in the smaller bronchi. An apparatus was introduced by Leonard Hill for use in collapsed and unconscious cases: a foot-pump feeds a face-mask through a flexible tube; by each downstroke a measured volume of air or oxygen is pumped into the lungs, and by each upstroke a valve is opened which allows the air to escape by the elastic recoil of the thorax and lungs. From time to time the fluid is evacuated by squeezing the thorax and hanging the head over the side of the stretcher.

Unless the patient is collapsed or unconscious, vomiting gives great relief by expelling large quantities of yellowish frothy fluid from the lungs; if this does not occur spontaneously, the patient puts his finger down his throat after drinking half a pint of warm salt water. Ipecacuanha and apomorphine should not be used.

The inhalation of oxygen relieves cyanosis and improves the patient's condition. But it is very difficult to get a patient

who is fighting for breath to tolerate any form of mask, without which it is impossible to give oxygen really efficiently. Administered in the ordinary way through an open funnel held near the patient's face the oxygen in the alveolar air is only increased by 1 or 2 per cent., whereas by using a mask and suitable apparatus the alveolar air should contain as much as 70 per cent. of oxygen.

Theoretically atropine should help to diminish bronchial spasm and secretion during the first twenty-four hours; but it has been found useless in severe cases and disappointing in slighter ones. Atropine is certainly valuable in the attacks of dyspnœa which may occur during convalescence, and I have found that stramonium taken regularly diminishes the liability to these attacks; potassium iodide in small doses is also useful.

Inhalations of ammonia are useful in the earliest stages, and after the second day ammonium carbonate in doses of gr. v every three hours produces copious expectoration, which results in improvement of colour and considerable relief. When great restlessness and mental distress are present, morphia should be injected.

Extreme cyanosis with a full pulse is greatly relieved by bleeding: breathing becomes easier, headache is relieved, and the patient falls into a refreshing sleep. Lian and Hebblethwaite found that the effect is most marked if venesection is performed in the first few hours. From 15 to 25 ozs. of blood should be slowly removed. The blood is dark and coagulates with abnormal rapidity. Bleeding is contra-indicated if the patient is pale and collapsed.

References

On the Treatment of Wounds in War, London, 1915.

Sir J. R. Bradford and T. R. Elliott: *British Journal of Surgery*, III. 234, 1915.

J. Elliott Black, E. T. Glenny, and J. W. McNee: *Brit. Med. Journal*, 1915, II. 165.

Leonard Hill: *Brit. Med. Journal*, 1915, II. 801.

A. S. Hebblethwaite: *Brit. Med. Journal*, 1916, II. 107.

E. Sergent and E. Agud: *Bull. et Mém. de la Soc. Méd. des Hôpitaux de Paris*, XXXIX. 960, 1915.

C. Lian: *Bull. et Mém. de la Soc. Méd. des Hôpitaux de Paris*, XXXIX. 1067, 1915.

INDEX

Abscess of liver, 64
Agarophobia, 13
Amblyopia, hysterical, 14
Amnesia, 9
Amœbic abscess of liver, 64
dysentery, 41
encephalitis, 44
hepatitis, 64
Anaphylaxis, 58
Aphonia, hysterical, 25
Appendicitis and amœbic dysentery, 47
paratyphoid fever, 99
trench fever, 77
Asthenopia, functional, 13

Bacillary dysentery, 52
Balantidium coli and dysentery, 61
Beri-beri, 116
and jaundice, 117
Blepharospasm, hysterical, 14
Blindness, hysterical, 7, 15
Bradycardia in epidemic jaundice, 111
paratyphoid fever, 92
Bronchitis in gas-poisoning, 145
Broncho-pneumonia in gas-poisoning, 145
paratyphoid fever, 94

Carbon-monoxide poisoning and shell-shock, 5
Cerebro-spinal fluid in shell-shock, 3
Cerebro-spinal meningitis and paratyphoid fever, 99
and trench fever, 76
Charcoal treatment of dysentery, 50
Chlorine poisoning, 142
Cholecystitis in epidemic jaundice, 110
paratyphoid fever, 95
Ciliate dysentery, 61
Cirrhosis of the liver, 68
Concussion and shell-shock, 3
Contractures, hysterical, 27
Convulsive symptoms in shell-shock, 26

Deafness, hysterical, 19
Diarrhœa: treatment during dysentery epidemics, 62
Disseminated sclerosis, 34
Double personality, 10
Dumbness, hysterical, 7, 21
Dysentery, 41
amœbic, 41
bacillary, 52
ciliate, 61
flagellate, 60

Emetine in amœbic dysentery, 49
hepatitis and hepatic abscess, 70
Encephalitis, amœbic, 44
Entamœba coli, 42
dysenteriæ, 42
histolytica, 42
Enteric fever: *vide* paratyphoid and typhoid fever, 88
Epidemic jaundice, 104
Epilepsy, 35
hysterical, 26

Fatigue in shell-shock, 11
Fever, paratyphoid, 88
trench, 71
typhoid: diagnosis from paratyphoid fever, 97
Fits, epileptic, 35
hysterical, 26
Flagellate diarrhœa and dysentery, 60
Flexner bacillus, 52
Functional nervous disorders (*vide* Hysteria), 1

Gas-poisoning, 142
General paralysis of the insane, 34

Hallucinations in shell-shock, 12
Headache and shell-shock, 11
Hearing, disorders of, in shell-shock, 19

Heart in epidemic jaundice, 127
paratyphoid fever, 91, 127
trench fever, 85, 127
irritable, 126
soldier's, 126
tobacco and, 127
Hemiplegia, hysterical: diagnosis from organic, 28
Hepatic abscess, 64
Hepatitis, amœbic, 64
Hyperacusis, 20
Hyperthyroidism, 130
Hypnotism, 36
Hysteria, 1
Hysterical amblyopia, 7, 14
aphonia, 25
blindness, 7, 14
contractures, 27
deafness, 19
dumbness, 7, 21
fits, 25
mutism, 7, 21
paralysis, 27
stammering, 24
tremor, 26
Hystero-epilepsy, 26

Influenza: diagnosis from paratyphoid fever, 98
from trench fever, 84
Insomnia in shell-shock, 12
Irritability in shell-shock, 11
Irritable heart, 126

Jaundice, epidemic, 104
and beri-beri, 117
dysentery, 107
paratyphoid fever, 95, 108

Lamblia dysentery, 60
Lead poisoning and nephritis, 136
Leucocytes in amœbic dysentery, 46
hepatic abscess, 66
paratyphoid fever, 97
trench fever, 77
Lice and trench fever, 75
Liver abscess, 64
cirrhosis of, 68
in epidemic jaundice, 111

Malaria: diagnosis from paratyphoid fever, 99
trench fever, 84
Malingering, diagnosis of, 33
Malta fever: diagnosis from paratyphoid fever, 98
Meningitis: diagnosis from paratyphoid fever, 99
from trench fever, 76
Micrococcus tetragenus septicæmia, 98
Mutism, hysterical, 21

Nephritis in gas-poisoning, 143
in paratyphoid fever, 94
in trench fever, 85
trench, 135
war, 135
and metal poisoning, 136
Nervous disorders, functional, 1
organic, 34
Neurasthenia: etiology, 2
Neuritis, peripheral and epidemic jaundice, 117
Nightmares, 12

Obsessions, 13
Ocular symptoms in shell-shock, 13

Pain, hysterical, 33
Paralysis agitans, 34
Paralysis, hysterical, 27
Paratyphoid fever, 88
and epidemic jaundice, 108
Periodic type of trench fever, 80
Peripheral neuritis and jaundice, 117
Peritonitis in paratyphoid fever, 95
Pneumonia in paratyphoid fever, 94
Poisoning, carbon-monoxide, 5
chlorine, 142
gas, 142
metallic and nephritis, 136
P.U.O. (A.), 72

Quinine and trench fever, 86

Relapsing fever: diagnosis from trench fever, 85

Saline treatment of bacillary dysentery, 56
Salonica fever, 72
Sciatica, 34
Serum treatment of bacillary dysentery, 57
Shell-shock, 3
Shiga's bacillus, 52
Shin pain in trench fever, 78
Soldier's heart, 126
Spasm, hysterical, 27

Speech disorders in shell-shock, 21
Spirochæta ictero-hæmorrhagica, 114
Spleen in epidemic jaundice, 111, 114
paratyphoid fever, 89
trench fever, 90
Stammering, hysterical, 24
Stupor and shell-shock, 8
Suggestion, 36

Tabes, 34
Tachycardia in paratyphoid fever, 94
soldier's heart, 130
Tics, 24
Tin poisoning and nephritis, 136
Tobacco and soldier's heart, 125
Tremor, hysterical, 26
Trench fever, 71
nephritis, 135
shin, 82
Trichomonas diarrhœa and dysentery, 60
Typhoid fever: diagnosis from paratyphoid fever, 97

Uræmia in war nephritis, 138

Vaccination for bacillary dysentery, 55
paratyphoid fever, 101
Vegetative neurosis, 130

War nephritis, 135

THE END

PRINTED BY WILLIAM CLOWES AND SONS, LIMITED LONDON AND BECCLES.

175 mm—